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Office of Administrative Law Judges
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Issue Date: 03 March 2003

In the Matter of	:	
	:	
LOIS J. COHEN,	:	
Claimant,	:	
	:	**Case No.: 2002-LHC-0368**
v.	:	**(formerly 2000-LHC-0246)**
	:	
PRAGMA CORPORATION/	:	**OWCP No.: 02-125596**
CIGNA INSURANCE CO.,	:	
Employer/Insurance Carrier, and	:	
	:	
DIRECTOR, OFFICE OF WORKERS'	:	
COMPENSATION PROGRAMS,	:	
Party-in-Interest.	:	

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For the Claimant

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For the Employer/Insurance Carrier

Before: PAMELA LAKES WOOD
Administrative Law Judge

DECISION AND ORDER GRANTING MODIFICATION AND BENEFITS

The instant case involves a request for modification relating to a claim brought under the Defense Base Act, 42 U.S.C. § 1651 ***et seq.***, an extension to the Longshore and Harbor Workers' Compensation Act ("the Act"), 33 U.S.C. § 901 ***et seq.*** Implementing regulations appear at 20 C.F.R. Parts 701 to 704. As in her original claim, Claimant Lois J. Cohen ("Claimant") claims to have sustained pulmonary fibrosis with multiple complications as a result of exposure to environmental pollutants in Almaty, Kazakhstan, where she was employed as an attorney by Employer Pragma Corporation, Inc. ("Employer") from November 1997 to May 1998. Employer is insured by Cigna Property and Casualty Insurance Company ("Carrier").

Following an October 27, 2000 hearing, Claimant's original claim for benefits under the Act was denied by the undersigned's May 24, 2001 Decision and Order Denying Benefits (which is incorporated by reference herein).[1] At the hearing, Claimant's Exhibits A through F, G1, G2, G3, H1, H2, and I1 through I7,[2] and Employer's Exhibits 1 through 4[3] were admitted into evidence. (TR1 at 9-12). Following the hearing, the record was left open for the transcripts of the depositions of Dr. Randall Wagner and Dr. Carl Friedman, which were admitted into evidence as Claimant's Exhibits J and K ("CX J" and "CX K"), and the record closed. Following briefing, the undersigned denied the claim, finding that although the Claimant had established a ***prima facie*** case, giving rise to the presumption under section 20(a) of the Act that she sustained some harm due to potentially causative working conditions, causation had been rebutted by Dr. Friedman's deposition testimony and she was unable to sustain her overall burden of persuasion. Claimant appealed the denial of her claim to the Benefits Review Board, and her appeal was assigned BRB No. 01-739.

On September 19, 2001, Claimant filed a timely motion for modification Accordingly, the Benefits Review Board dismissed her appeal, subject to reinstatement, and remanded the case to the District Director for modification proceedings. Following processing, on November 14, 2001, the District Director transmitted the instant case to the Office of Administrative Law Judges for a hearing.

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[1] The transcript of the first hearing before the undersigned, which took place on October 27, 2000, will be referenced as "TR1" followed by the page number.

[2] Claimant's Exhibits A through F from the first hearing appear in Binder A at pages 4 through153. Binders G1 (with subparts A and B), G2 (with subparts C, D, and E), and G3 (with subparts F, G, and H) consist of Claimant's Medical Treatment Records (with George Washington University hospital records appearing in Binder G1 at pages 157 through 717; medical records from Drs. Randall Wagner, James H. Graeter, and Allen Greenlee appearing in Binder G2 at pages 721 through 921, with inserted pages 784B and 785A; and medical records from Dr. Richard Edelson, Vienna Medical Records, and Dental Records appearing in Binder G3 at pages 925 through 1032). Binder H1 consists of an itemization of medical bills and receipts, appearing at pages 1050 through 1433 (with summaries at pages 1034 through 1049). Binder H2 consists of supplemental exhibits, including photographs, additional dental records, Claimant's February 19, 1999 and April 5, 1999 notices of claims, Employer's April 9, 1999 acknowledgment of claim, Claimant's April 12, 1999 Form LS-203 claim, and Employer's April 21, 1999 Form LS-207 controversion, appearing at pages 1436 to 1482. Claimant's Exhibits I1 through I7 are photographs.

[3] Employer's Exhibits 1 and 2 ("EX 1" and "EX 2") from the first hearing are Dr. Carl B. Friedman's July 30, 2000 report (pages 1 through 7) and Dr. Friedman's curriculum vitae (pages 8 through 13), respectively; Employer's Exhibit 3 (page 14) ("EX 3") is Claimant's registration form with Dr. James H. Graeter; and Employer's Exhibit 4 (pages 15 to 33) ("EX 4") consists of medical records from Dr. Arthur I. Kobrine.

A hearing relating to the modification request in this matter was held before the undersigned administrative law judge on July 22, 2002, in Washington, DC.[4] The record from the previous hearing was obtained by the undersigned from the Director, Office of Workers' Compensation, and I granted the parties' joint request that the entire record from the previous proceedings be considered. (TR2 at 5 to 6.) In addition, Claimant's Exhibits L1 through L34 and Employer's Exhibits E1 and E2 were admitted into evidence at the hearing.[5] (TR2 at 36, 219-23). The Claimant and her treating physician Dr. Randall P. Wagner (who also acted as an expert witness) were the only witnesses to testify at the hearing; however, Employer's expert witness, Dr. Carl Friedman, testified by deposition. (TR2 at 22 to 60, 61 to 219; EX E2). At the end of the hearing, the record closed, except that the Claimant agreed to submit an affidavit explaining the sources of the articles comprising Claimant's exhibits. Briefs or written closing arguments were to be submitted within 60 days of the hearing date. Both Claimant and Employer/Carrier submitted timely briefs, which were filed on September 23, 2002 and September 24, 2002, respectively.[6] Along with her brief, Claimant submitted the Affidavit of Elaine Shepard, Office Administrator and research assistant at Claimant's counsel's law firm, which I have marked as "ALJ 1" and which is hereby admitted into evidence. **SO ORDERED.**

The findings and conclusions which follow are based upon a complete review of the record in light of the submissions of the parties and the applicable statutory provisions, regulations, and pertinent precedent.

STIPULATIONS

The parties adopted the Stipulations they reached at the previous hearing, set forth below:

1. There was an employer/employee relationship between Claimant Lois Cohen and Employer Pragma Corporation at the time of her alleged work injury.

2. There is jurisdiction under the Defense Base Act and Longshore Act over the instant claim.

3. Employer was provided timely notice of this claim.

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[4] The transcript of the second hearing before the undersigned – the July 22, 2002 hearing – will be referenced as "TR2" followed by the page number.

[5] The exhibits at the second hearing are variously referenced as "Employer's Exhibits 1 and 2" and "E-1" and "E-2", but herein they will be referenced as Employer's Exhibits E1 and E2 ("EX E1" and "EX E2", respectively) in order to avoid confusion with the Employer's Exhibits 1 through 4 ("EX 1" through "EX 4") received in evidence at the previous hearing. (TR2 at 221-22).

[6] Although the original of the brief was complete, the copy was missing two pages, which Claimant submitted under cover letter of September 26, 2002.

4. Claimant's average weekly wage exceeds the maximum allowable average weekly wage for purposes of this worker's compensation claim under the Longshore Act/Defense Base Act.

5. Claimant filed a timely claim.

6. Claimant fully cooperated in the resolution of this claim.

(TR 2 at 10 to 11; CX B; TR1 at 34, 43.)

ISSUES

There is an initial threshold issue of whether the Claimant has established a change in conditions or mistake in determination of fact so as to give rise to modification under the Act. ***See*** 33 U.S.C. § 922.

If that initial hurdle is overcome, the case will be reopened, and it must again be determined whether Claimant's pulmonary fibrosis may be deemed to have arisen out of the course of her employment in Almaty, Kazakhstan, by operation of the presumption under section 20(a) of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 920(a). If so, I must determine whether Employer has rebutted the presumption. If rebuttal is established, I must address the issue of causation based upon all of the evidence.

Again, in the event that Claimant can establish a causal link between her employment and her pulmonary fibrosis, additional issues concern whether Claimant has established that her multiple complaints (including a right hip condition, shingles (herpes zoster), ulnar nerve neuropathy, coronary artery disease, carbon stent implant, facial disfigurement ("moon face"), a left hip condition, and dental tooth decay and disease) were related to or aggravated by either the pulmonary fibrosis or Prednisone treatment for the pulmonary fibrosis. Claimant is claiming medical benefits based upon her expenses for the treatment of these conditions and she is also claiming compensation for periods of temporary total disability (TTD) necessitated by them.[7] (Claimant's Pre-hearing Statement at 1, 4, 5). The TTD periods claimed consist of June 6, 1998 to February 1, 1999 for pulmonary fibrosis and partial lung biopsy, and for right hip surgery; March 3, 2000 to March 21, 2000 for left ulnar nerve surgery; May 14, 2000 to June 8, 2000 for carbon stent implant in the heart; and October 27, 2000 to June 1, 2001 for left hip replacement surgery. (Claimant's Pre-Hearing Statement; ***see also*** TR2 at 43 to 44).

[7] Although the transcript shows that Claimant at one point indicated there was no TTD claim, contrary to other statements, my recollection is different. I believe there was a mistranscription. (***Compare*** TR 2 at 46 ***with*** TR2 at 11).

The issue of whether Claimant is permanently disabled, on a total or partial basis, is not before me at this time. (TR2 at 44-46).[8]

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Summary of Facts from Previous Decision

The evidence before me at the prior decision and the facts that could be gleaned therefrom were summarized in the body of that decision, which I have incorporated verbatim herein (except as otherwise indicated).

Claimant's [October 27, 2000] Testimony

Claimant was a credible witness. She testified that she was born on December 7, 1941 and was 58 years old at the time of the hearing.[9] (TR1 at 47). She graduated from Emory Law School in August 1983 and, following eight years of work in the brokerage area, she started doing what she described as "this international kind of work." (TR1 at 47). Her first assignment was in Kiev for three months in late 1996, after she returned home for a couple of months she went to Bulgaria for seven months, and after she came home from that she left for Kazakhstan for a 12 to 15 month assignment. (TR1 at 47-48). She testified that prior to November 1997, when she began working for Pragma Corporation (based in Falls Church, Virginia), she was in good health, except that she started having pain in her right leg in 1996, which led to disc surgery in July 1996. (TR1 at 48, 51). She was assigned to work in Kazakhstan as a Senior Legal Advisor to the Securities Commission that had recently been established in that country. (TR1 at 49). Initially, she was hired to be their advisor in Kyrgyzstan, and after a couple of days in Almaty she was transferred to Kyrgyzstan, where she worked for two weeks. (TR1 at 50). At that point she was transferred back to Almaty, which is about a three hour drive away. (TR1 at 50).

Claimant testified that as they approached Almaty, she noticed a "huge black cloud, like you couldn't see through" and when she asked the driver what it was, he said, "That's Almaty." (TR1 at 50). She testified that it was only later that she learned about "leftover nuclear problems and radiation and pesticides, pollution." (TR1 at 50).

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[8] According to Claimant's counsel, the issue of "whether or not she will be able to return to work and whether or not her inability to return to work is related to her inability to tolerate the work arising from what has happened to her in Almaty, Kazakhstan" should be left open until Claimant could be examined, and she may be entitled to additional benefits on that basis. (TR 2 at 45).

[9] Claimant's stated age was mistranscribed as "38." (TR1 at 46).

Claimant first developed symptoms in late March or early April, 1998, when she started to have chronic diarrhea. (TR1 at 50-51). After a couple of weeks she contacted the American doctor there (Dr. Bassett) who attributed it to "the water or the food or whatever" and he gave her an antibiotic which helped, but as soon as she went off the antibiotic, which she received as part of 10-day treatments, the diarrhea started again. (TR1 at 51, 53). At that point, the doctor said he could not culture what she had and even if he could, he would not have the drugs to treat it, so at the end of April he recommended that she return home. (TR1 at 51). In retrospect, she realized that she was also experiencing pulmonary problems. (TR1 at 51). When her friend arrived around April 1 to work with her, they used to go swimming every night at her hotel, and she noticed difficulty swimming the 10 to 20 laps, and she would be dizzy when she got out of the pool (TR1 at 51-52). When her friend got off the plane, the friend told her that she looked yellow. (TR1 at 52). She also noticed herself going slower and slower when walking up stairs, which she took to her apartment and to the third or fourth floor of the U.S. A.I.D. mission where she worked, and she took the elevators whenever she could. (TR1 at 52). She attributed the problems to her leg. (TR1 at 52). However, she indicated that she also experienced shortness of breath, which was "really bad" while swimming. (TR1 at 52). Although the head of the U.S. A.I.D. mission did not want her to leave, the project director (Mohammed [Fatoorechie]) allowed her to leave due to her illness, and she finally got on a plane in May. (TR1 at 53). She was supposed to return to Kazakhstan two weeks later, as she was accustomed to do after visiting her daughter and granddaughter, which she did approximately every three months. (TR1 at 53-54).

Upon her return, Claimant saw her internist, Dr. Greenlee [mistranscribed as Greenly] for "horrible cystitis" as well as the diarrhea, and she was "taking stool cultures every day." (TR1 at 54). In late May of 1998, Dr. Greenlee referred her to Dr. Moscowitz due to her leg pain, and she had her hips x-rayed. (TR1 at 54-55). Dr. Moscowitz told her that she needed hip surgery because "the right hip is going" but that she could wait until her assignment in Kazakhstan was finished. (TR1 at 55).

One Saturday (June 6, 1998), when her other daughter [Laura] from Atlanta came to visit her and Claimant went to the airport to pick her up, Claimant was coughing and could not catch her breath, and she went to visit the emergency room at GW [George Washington University] Hospital. (TR1 at 54). She complained that she could not breathe and she was put on oxygen. (TR1 at 55). She does not remember the next 12 days, aside from some pain when they were trying to get blood. (TR1 at 56). When she awoke, all three of her daughters were at the hospital, which made her realize it was serious. (TR1 at 56). She met Dr. Randall Wagner, a pulmonary specialist, shortly afterwards, and he became her treating physician. (TR1 at 57-58). Dr. Wagner told her that her condition, pulmonary fibrosis, was not caused by cigarette smoking. (TR1 at 58).

After the lung biopsy, Dr. Wagner put her on very high-dose Prednisone, from 80 mg.[10] for two months, then 60, then 40, and then 20, right before her hip surgery. (TR1 at 58-59). It was her understanding that she was put on Prednisone because the lung biopsy showed scarring and inflammation on her lungs. (TR1 at 59). It was also her understanding that the reduction in dose was made because Dr. James Graeter [mistranscribed as Grader], the orthopedic surgeon, would not operate on her if she were on a higher dose, because the Prednisone would destroy what was left of the hip. (TR1 at 59-60).

After her discharge from the hospital, Claimant was sent to a full-time care facility for a week, where she continued on Prednisone. (TR1 at 61). She discharged herself and retained full-time nursing at home. (TR1 at 62).

After she was home for a week, she experienced excruciating pain going all the way down her left arm, beginning at her neck, followed by boils extending down the arm, and the nurse told her that she had shingles. (TR1 at 63-65). She has some scarring on that arm, and at the hearing I observed a depigmented area of scars on the upper left arm, extending down the forearm. (TR1 at 64). Dr. Wagner gave her a morphine (Fetinol) patch for the pain. (TR1 at 65).

In September or October of 1998, she experienced dental problems and she started having pain in her gums where her teeth had been capped. She also observed separation from the gums. (TR1 at 65). In September and October 1999, her dental problems worsened, shortly before she was supposed to leave for Sarajevo. (TR1 at 76). The teeth that Dr. Nielsman had done the previous year had rotted out and she needed to have root canals, causing her to lose four of the eight teeth on her bottom jaw and requiring the whole bridge to be redone. (TR1 at 76-77). She is still experiencing mouth and dental problems, involving the top of her mouth and ten or eleven teeth. (TR1 at 82).

She also had the right hip surgery [in October 1998], which consisted of replacing her hip with metal. As she understood it, "the cortisone had really destroyed whatever was left in the hip or [her] ability to walk, and it was crucial to all the doctors that [she] be able to start walking around and getting a little exercise." (TR1 at 66). She returned home and had to hire nursing care, because she could not go down the stairs although she could walk with a walker. (TR1 at 66). It was her understanding that Dr. Graeter has diagnosed her as temporarily totally disabled due to her hip, because she cannot walk very far. (TR1 at 82).

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[10] I could not find a reference to 80 mg. of Prednisone in the medical records. See the Routine Medication Record, CX G1, p. 176, 624 (showing Prednisone dose of 60 mg. daily from June 15 to 18, as ordered on June 15, 1998) and the June 18, 1998 discharge summary CX G1, p. 466 (showing a Prednisone dose of 60 mg. for 6 months).

When Claimant woke up from the hip surgery, the shingles (herpes zoster) pain was gone, but it was replaced with "horrible itching." (TR1 at 69). She went to see Dr. Edelson, a neurologist, who put her on Neurontin, which she took daily over a two-year period, tapering down shortly before the hearing, although she was still taking it. (TR1 at 69, 76). She is now taking one or two Dilantin a day instead of six. (TR1 at 89). The shingles blew up again when she was in Georgia, and she was treated with Celebrex or some other anti-inflammatory. (TR1 at 88-89). However, she continued to have problems with numbness in that arm and hand, leading to ulnar nerve surgery in February 2000, when she temporarily came home from Bosnia, returning in approximately mid-March. (TR1 at 77-78).

While in Bosnia, Claimant experienced heart problems, first noticing pain and pressure in her heart around May 14 [2000] (Mother's Day), continuing nightly for a week and making it difficult for her to breathe. (TR1 at 79). She was airlifted out of the hospital after about five or six days, and she had an angiogram in Vienna, where she was hospitalized for four days, with a stint implanted. She was required to wait two to two and one half weeks before she could travel back to Sarajevo, which she did around June 6, 2000. (TR1 at 79-80).

Claimant had finally gone off Prednisone therapy in mid-February [1999], and at that time she left to go to Latvia, as a legal advisor concerning the stock exchange, because she had bills to pay. (TR1 at 68, 88). At that time, she first learned about the Defense Base Act. (TR1 at 69-70). Pragma employees never told her about those benefits, prompting her to write to Mohammed Fatoorechie on February 19, 1999; her letter appears at CX H2, p. 1468-69. (TR1 at 70-71). She came back from Latvia in April 1999. (TR1 at 75-76). Her next job overseas was for Barron's Group in Tbilisi, Georgia, for three months, beginning in mid-June 1999, doing the same kind of work. (TR1 at 76). Her last assignment was in Sarajevo, Bosnia, beginning on October 1,1999, where she worked as a Chief of Party for a large project which involved establishing a securities commission, for a year (as extended). (TR1 at 78-79, 90-91). Her work in Sarajevo did not require anything physical, as a driver took her to work and it was a desk job. (TR1 at 95-96). In all, Claimant has been unable to work 14 months out of the past 28 months, since her return from Kazakhstan. (TR1 at 82).

Claimant has also noticed a difference in her physical appearance, as reflected by seven photographs appearing in CX I7 (taken in June 1996), CX I6 (taken in November or December 1996), CX I5 (taken in July 1997), CX I4 (taken in December 1998), CX I2 and I3 (pre-surgery photographs taken in June 1999) and CX I1 (taken following plastic surgery), as well as CX H2, pages 1437 through 1438 (taken in December 1998) and CX H2, p. 1439 (taken in September 1999, after the plastic surgery.) (TR1 at 74-75). Claimant had plastic surgery in early May, 1999. (TR1 at 75).

At the time of the hearing, Claimant complained of her left hip pain and indicated that she could barely walk and will require surgery. She had not previously had problems with the left hip. (TR1 at 81). As far as her breathing is concerned, she will think she is breathing fine but will be unable to walk up a hill or climb stairs, particularly if she has to lift anything heavier than three pounds. (TR1 at 81). She plans to have dental surgery and left hip surgery when she has the money to pay for it. (TR1 at 92-93). The hip surgery was scheduled for October 30 and was canceled. (TR1 at 92). The surgery would be covered by health insurance, but she is concerned about the need for nurses for six weeks after the surgery. (TR1 at 94-95).

Claimant verified that the medical bills in Binder H1 reflect her out of pocket expenses for medical care she has received since her return from Kazakhstan in late May 1998. (TR1 at 82-83; ***see also*** CX H1).

Claimant does not feel that she is now capable of returning to work and she expressed concern about the availability of treatment for her heart problems overseas, including Sarajevo, where they lacked the capability of checking her cholesterol monthly. (TR1 at 83-84). She believes both her hip and her heart are disabling, but the dental condition is not. (TR1 at 93). Claimant feels that her hip condition has deteriorated from September 9 until the date of the hearing, October 27, 2000. (TR1 at 95). If she had a driver such as she had in Sarajevo, she could probably do the legal work she was doing, but she is concerned about her heart. (TR1 at 96).

On cross examination, Claimant admitted to having been on several different drugs on May 28, 1998, including low-dose Prozac for pain, Voltarin as an anti-inflammatory, Percodan for pain, and hormone replacement therapy. (TR1 at 86-87). They were prescribed by Dr. Greenlee or Dr. Charott, the pain management doctor. (TR1 at 86). She took the Voltarin for right leg pain. (TR1 at 87). Claimant still smokes cigarettes, having smoked on and off for forty years (less four years when she was on Proloxil and quit), at up to a pack a day, but she is down to half a pack. (TR1 at 87).

Claimant had a chest x-ray in June 1996, before they did the back surgery, and it was her understanding that it was normal. (TR1 at 97). She may have had a physical before her first job, in Kiev. (TR1 at 97).

Medical Records and Reports

Numerous medical records and other documents have been received into evidence, as noted above, and only ones of particular significance will be discussed here.

Records Predating Hospitalization

Medical records of Claimant's treating physician, **Allen Greenlee, M.D.** predate her hospitalization for pulmonary fibrosis in June 1998. (CX G2, p. 824 to 921). Dr. Greenlee's records also relate to Claimant's treatment after her discharge from the hospital (***Id.***, p. 825A, 826, 829-32, 841, 843, 847, 887, 889, 902, 907, 911).

Dr. Greenlee's records relating to Claimant begin in January 1996, when a herniated disc was noted, and include copies of records relating to her surgery (diskectomy) in July of the same year and subsequent treatment for back pain, including epidural nerve blocks. (***Id.*** at 867-79, 885-886). In a progress note of August 1996, it was noted that the Claimant was on Diflunisol (?), Prevacid, and Prempro, that she had elevated cholesterol, and that she smoked approximately one pack of cigarettes per day. (***Id.*** at 878). Dr. Greenlee gave the Claimant a flu shot in October 1997 (prior to her departure for Kazakhstan) and noted that she was on various medications (Percodan, Prozac, Prevacid, Vitron, Prempro, Valium, and Darvocet).[11] (***Id.*** at 920). The note of February 9, 1998 (apparently when she was on leave from Kazakhstan), which is partly illegible, recorded that she had complained of increased back pain and an epidural block was considered, but that Claimant was going back to Eastern Europe. Although not entirely legible, the note also mentions something about Claimant wanting a hepatitis A shot. (***Id.*** at 920).

A note of May 26, 1998 (appearing in Dr. Greenlee's medical records but signed illlegibly) reflects that the Claimant presented to the clinic with multiple complaints: (1) diarrhea accompanied by severe epigastric pain, bloating, and increased flatulence dating from approximately two months before in Kazakhstan, which condition was treated successfully with unknown medications but returned upon discontinuance of the medications; (2) back pain with a shooting pain down the left leg, for which Claimant requested a renewal of her Percodan prescription; and (3) chronic cystitis, with dysuria and polyuria. A stool culture and urine culture were planned; Claimant was referred to Dr. Moskovitz and Dr. Cherrick for the back pain and sciatica; and Claimant was prescribed Prevacid (?) for the gastrointestinal problems and Pyridium for the cystitis. (***Id.*** at 864, 890). The urine culture report of May 28, 1998 was negative for pathogens and the stool culture report of June 3, 1998 found no ova and parasites and no salmonella, shigella, or campylobacter was isolated. (***Id.*** at 861-63).

Arthur I. Kobrine, M.D., a neurological surgeon, treated the Claimant in 1996 for right lumbar radiculopathy and performed a right lumbar laminectomy and diskectomy on July 18, 1996. He also treated her for acute lumbar strain following a slip and fall in August 1996 and for right leg/groin/knee pain. (EX 4).

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[11] Claimant had been on estrogen (Prempro) since 1995. (CX G2, p. 886, 896, 921). The records also reflect that Percodan and Prozac were prescribed for back pain. (***Id.***, p. 868, 920.) However, some of the medicine names are barely legible. ***Id.***

George Washington University Medical Center

Claimant was admitted to the **George Washington University Medical Center** on June 6, 1998 and discharged on June 18, 1998. (CX G1, p. 457-672; 159-198). According to the discharge summary dictated by **Trang Do, M.D.** and signed by **Allen Greenlee, M.D.**, the discharge diagnoses were usual interstitial pneumonitis with pulmonary fibrosis, degenerative joint disease of the hip, diarrhea, and anemia. (***Id.*** p. 464-67.) A computer listing indicated final diagnoses of postinflammatory pulmonary disease (principal diagnosis) as well as respiratory complication, anemia, primary localized osteoarthritis of pelvic area/thigh, and diarrhea. (***Id.*** p. 457). The discharge summary indicated the following history:

> HISTORY OF PRESENT ILLNESS: The patient is a 56-year old white female with no significant past medical history presenting to the emergency room complaining of 1-week history of upper respiratory infection with a nonproductive cough and shortness of breath of 2-3 days' duration. The patient recently returned from Russia after a 7-month stay for evaluation of chronic diarrhea. While in Russia, the patient reports shortness of breath and dyspnea on exertion initially attributed to her bouts of diarrhea. Then 2-3 days prior to her admission, she developed worsening shortness of breath, subjective fevers and chills, and a nonproductive cough. The patient reports that her diarrhea has resolved since her return to the United States for the last 2 weeks.
>
> PAST MEDICAL HISTORY:
>
> 1. Chronic diarrhea, evaluation in Russian was nonrevealing; however, each bout resolved with metronidazole.
> 2. Chronic lower back pain, recently diagnosed with arthritis of the right hip requiring total hip replacement. She is status post surgery on her back at L3 and L4.

(***Id.*** p. 464-66). A 40-pack-year smoking history was noted, as was an allergy to penicillin. (***Id.***). A bronchoscopy biopsy was inconclusive, leading to an open-lung biopsy of the right lower lung which "was consistent with the usual interstitial pneumonitis with pulmonary fibrosis." (***Id.; see also*** p. 548-50, 713-15.) The diagnosis on the June 12 surgical pathology report for the June 10, 1998 wedge biopsy[12] by **Arnold M. Schwartz, M.D.** was "active and organizing interstitial pneumonitis consistent with exudative and proliferative phase of diffuse alveolar damage (DAD)" and it was noted that the routine

[12] Although the pathology report states that the specimen was taken on June 11, 1998, the surgery was performed on June 10 according to the operative report. (CX G1, p. 549-52).

stains had identified no granulomas, fungi or viral inclusions." [13] (CX G1, p. 548-50, 713-15). At that point, the Claimant was treated with high dose Solu-Medrol for three days and then switched to Prednisone.[14] (***Id.*** p. 464-66, 176). Medications on admission were noted to be Prozac, Percodan, Prevacid, Prempro, and Valium, and the discharge medications were the same with the addition of Prednisone at a dose of 60 mg. orally per day, to be continued for approximately 6 months. (***Id.*** p. 464-66).

In a report of July 10, 1998, **Randall Wagner, M.D.** noted that he had seen the Claimant that date in follow up for her interstitial lung disease, which he characterized as "Stable/improving interstitial pneumonitis." He recommended continuing the Prednisone at 40 mg. per day and reducing it to 30 mg. per day if the pulmonary function tests were good. He also recommended a hip replacement after three months of therapy. It was noted that Claimant had developed vesicular lesions on her left arm in the antecubial space and was started on famciclovir for Herpes Zoster, and that she tolerated the medication poorly with anorexia and nausea, but that the condition was resolving. It was also noted that she had not returned to smoking. (CX G1, p. 716-17).

Progress notes indicate that Claimant was treated on an outpatient basis at the Ambulatory Care Center at George Washington University Hospital on July 10, 1998, August 14, 1998, and September 25, 1998 (when her Prednisone was decreased to 20 mg.) (CX G1, p. 691-93). An October 2, 1998 letter from Dr. Wagner to Dr. James Graeter noted that she had Idiopathic Pulmonary Fibrosis and had "received 2 months of Prednisone 60-40 mg day, and has been tapered to 20 mg daily over the past month," noted bibasilar fine crackles on examination, and indicated that she was "as ready as she will ever be for the hip replacement." (CX G1, p. 690).

Discharge summaries from the George Washington University Medical Center of October 19, 1998 and October 23, 1998, by **James Graeter, M.D.** and **Philip Marion, M.D.**, respectively, reflect that Claimant had a history of idiopathic pulmonary fibrosis diagnosed in June 1998 by lung biopsy, that a total right hip replacement (due to right hip degenerative joint disease) was performed on October 14, 1998, that she was transferred to rehabilitation services on October 19, and that she was discharged on October 23. In the latter discharge summary, it was noted that she had a history of L1-2 discectomy in 1996 and chronic lower right extremity pain for a number of years, a history of half a pack of tobacco for one year (having quit three months previously), and no history of alcohol abuse. (CX G1, p. 306-07, 201-02; ***see also id.*** p. 340-42 [operative report by Dr. Graeter]). The October 14, 1998 ICU admission note by **Michael S. Salem, M.D.**, indicated that Claimant "has had multiple intensive care unit stays for this pulmonary

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[13] Tests for tuberculosis (PPD and cultures) were also negative. (CX G1 p. 481-83, 575-76.)

[14] The first Prednisone order of record is dated June 15, 1998. (CX G1, p. 176, 624; ***compare*** p. 465-66).

fibrosis, and is always on the border of requiring significant pulmonary intervention", that she was on home oxygen therapy, and that she was "critically ill." (***Id.*** p. 333). The transfer summary of the same date reflects that she was being treated, ***inter alia***, with Prednisone at an oral dose [po] of 20 mg. daily [qd]. (***Id.*** p. 330; ***see also id.*** p. 264, 398-99, 423, 445).

George Washington University outpatient notes reflect that Claimant was seen on an **outpatient** basis on October 30, 1998, November 2, 1998 (when she was admitted to the emergency room), December 4, 1998, and January 28, 1999, in addition to a telephone conversation on January 8, 199[9].[15] (CX G1, p. 684-89.[16])

Claimant was admitted to the **emergency room** for shortness of breath on November 2, 1998 and discharged the following day. (***Id.*** p. 425-56, 685-88). A November 2, 1998 lung ventilation and perfusion study by **Carmen R. Britt, M.D.** was normal, with no evidence of pulmonary emboli. (***Id.*** p. 239). As discussed below, a chest x-ray was within normal limits. (***Id.*** p. 433, 449-50). Prednisone dose was 20 mg. on November 3, 1998. (***Id.*** p. 453).

Following that admission, the Claimant's Prednisone dose was reduced. A clinic note of December 4, 1998 by **Dr. Wagner** listed the impression of "Stable usual interstitial pneumonitis, now on a tapering dose of steroids" and indicated that if the DLCO (diffusing capacity) remained in the 40% of predicted range, the Prednisone could be lowered to 10 mg. per day and they could "begin in earnest to taper her steroids." (***Id.*** p. 683). The January 28, 1999 note prescribed Prednisone at 5 mg, then 2.5 mg. (***Id.*** p. 679). Dr. Wagner's January 29, 1999 report indicated that Claimant was in the final stages of tapering off the Prednisone, that she had stabilized with a DLCO at about 48% predicted, and that she was off supplemental oxygen and gradually increasing her exercise. (***Id.*** p. 680).

Additional records from George Washington University Hospital relate to Claimant's treatment in 1999. They indicate that she was seen on March 29, 1999 for acute interstitial lung disease by **Dr. Morgan Delaney**, who noted that the Claimant's improvement with Prednisone raised doubts as to whether she had UIP, noted that she was "now off the steroids and says she feels well" although she wheezed when she coughed or laughed, opined that the UIP "has not followed the typical clinical course, and this raises the question whether this is the correct diagnosis", and suggested that she might have asthma. (CX G1, p. 677, 678.) **Chest x-rays** taken on April 7, 1999 were consistent with pulmonary fibrosis, as discussed below.

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[15] Although the reference is to "1/8/98", it is clear from the reference to "12/9/98" pulmonary function tests that 1999 is the year intended.

[16] There does not appear to be a page 688 of record in CX G1.

Claimant was treated on an outpatient basis by **Dr. Aamodt** for Dr. Delaney on September 13, 1999, when she was noted to have complained of dyspnea to Dr. Greenlee in New York the preceding week and it was noted that she was "still smoking." (CX G1, p. 676.)

Pulmonary function test reports appear for June 17, 1998 (with efforts technically inadequate to interpret data); August 11, 1998 (showing reduction in diffusion capacity but improvement in total lung capacity); September 21, 1998 (showing reduced diffusion capacity and terminal air flow); December 9, 1998 (no printed interpretation); April 7, 1999 (showing reduced diffusing capacity); [date obscured][17] (mild obstructive ventilatory defect without significant bronchodilator response, reduced diffusion capacity, normal arterial blood gas). (CX G1, p. 711-12, 709-10, 706-07, 704-05, 695-96, 697-98.; ***see also*** p. 679 (summary).)

Chest x-rays were taken during various hospitalizations and outpatient visits. An x-ray taken on June 8, 1998 was interpreted by **Jay M. Feder, M.D.** as showing "bilateral mixed pulmonary interstitial and air space disease, unchanged," one taken on June 9 was interpreted as showing "[i]nterval improvement in pulmonary interstitial edema" but "[p]ersisting diffuse bilateral air space disease"; and one taken on June 10, 1998 was interpreted by Dr. Feder as showing a "decrease in density of the lungs bilaterally consistent with resolving inflammatory process" and "[i]nterval improvement in edematous or inflammatory change within the lungs bilaterally". (CX G2, p. 891, 894, 917-19). An x-ray taken following the lung biopsy on June 10, 1998, was interpreted by Dr. Feder as showing "[b]ilateral pulmonary interstitial changes" "with interval increase from earlier"[18] and the impression was "[s]tatus post open lung biopsy with insertion of right chest tube and "[i]nterval increase in pulmonary interstitial edema," and Dr. Feder made similar findings (although noting more hypoventilation) on the June 11, 1998 x-ray (CX G1, p. 585-86). A chest x-ray taken at admission to the Emergency Room on November 2, 1998 noted clear and well expanded lungs without evidence for focal air space disease and the impression by **Sathy V. Bhaven, M.D.** and **Barry M. Potter, M.D.** was that there was "[n]o acute disease." (***Id.*** p. 449-50).[19] A chest x-ray taken on November 30, 1998 was interpreted by **Nishita N. Kothary**, M.D. and **Kaaren N.**

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[17] The values are the same as the December 1998 test, but the weight listed in almost 30 pounds off. (***Compare*** p. 704 ***with*** p. 697).

[18] [Footnote omitted. The reference is probably to the pre-biopsy x-ray interpretations of June 8, 9, and 10, 1998).]

[19] No fibrosis was noted on the two November 1998 x-ray interpretations. However, the April 7, 1999 x-ray report made findings of interstitial markings consistent with pulmonary fibrosis and indicated that November 30, 1998 films were reviewed, but did not comment upon any change in findings (CX G1, p. 449-50, 694, 699; CX G2, p. 897).

Bergquist, M.D. as showing [“]no evidence of air space disease, pleural effusion or pneumothorax” and “[n]o acute disease.” CX G2, p. 897). Chest x-rays taken on April 7, 1999 were interpreted by **Jocelyn A. Simon, M.D.** and **Edward M. Druy, M.D.** as showing [“]coarse interstitial markings identified bilaterally within the bases” “consistent with the patient’s known diagnosis of pulmonary fibrosis.” (CX G1, p. 694, 699.)

Additional Treatment Records

James H. Graeter, M.D. and **Peter A. Moskovitz, M.D.,** orthopedic surgeons, treated the Claimant for her back, right and left hip, and left arm conditions. (CX G2, p.784-823). Dr. Graeter performed a right hip total arthroplasty on October 14, 1998 and a left ulnar nerve transposition on March 3, 2000. (***Id.***, p. 786-88, 811).

Richard Edelson, M.D., a neurologist, treated the Claimant for her herpes zoster (shingles) and complications relating to its treatment. (CX G3, p. 925-36).

Records from **Vienna General Hospital** relate to Claimant’s treatment for a heart cond[i]tion. (CX G3, p. 937-1024).

Records from **Bruce Milzman, D.D.S.** relate to Claimant’s dental treatment. (CX G3, p. 1025-32).

Medical Opinion of Randall P. Wagner, M.D.

As noted above, **Randall P. Wagner, M.D.**, who is board-certified in internal medicine and the subspecialties of pulmonary diseases and critical care medicine, treated the Claimant for her pulmonary condition. (CX G2, p. 721-84; CX J Ex. 1). He is also the Claimant’s expert witness and his de bene esse deposition was taken on October 26, 2000. (CX J). At his deposition, he stated his opinion that the Claimant had idiopathic pulmonary fibrosis attributable to her exposure to environmental pollutants in Kazakhstan. (***Id.***)

Dr. Wagner noted that by history Claimant’s decline in respiratory status began in February or March of 1998 while she was in Kazakhstan, as reflected by her inability to climb the stairs to her apartment, but that she compensated by lifestyle modification (e.g., she used the elevator). (CX J, p. 13, 95). Dr. Wagner also noted that she had an intercurrent diarrheal illness but opined that there was no relationship between that disease and Claimant’s pulmonary fibrosis. (***Id.***) In Kazakhstan, she was treated for her diarrhea with Flagyl (Metronidazole), but he did not know what else Dr. Basset gave her. (***Id.*** p. 94). When she returned to the United States, her pulmonary symptoms continued to progress, bringing her to George Washington, where she was treated by Dr. Wagner. At that time, she had diffuse air space opacities, leading to complete respiratory failure and its treatment with Prednisone, with its resultant complications, until March 1999, when the

Prednisone was stopped altogether. (***Id.***, p. 14). The initial dose was three days of 1,000 milligrams per day, which is "organ transplant rejection doses." (***Id.***, p. 19). At the time Claimant was treated, the standard for treatment of idiopathic pulmonary fibrosis or cryptogenic fibrosing alveolitis was "six months of a milligram per kilogram of Prednisone with a taper at that point." (***Id.***, p. 18-19). When Claimant was down to 20 milligrams per day, Dr. Greenlee agreed to operate on her hip, which had progressed "quite quickly" when she was on the Prednisone, because at a higher dose, there would be delay wound healing with increased rates of infection, as well as loss of calcium. (***Id.***, p. 20-24).

Initially, Dr. Wagner diagnosed interstitial fibrosing pneumonitis, which is the same illness as pneumoconiosis, cryptogenic fibrosing alveolitis, and usual interstitial pneumonia. (***Id.*** p. 17). Fibrosis means scarring due to collagen deposition. (***Id.*** p. 32). The scarring is permanent. (***Id.*** p. 33).

On cross examination, however, Dr. Wagner conceded that Claimant would no longer qualify under the current ATS guidelines for usual interstitial pneumonia (UIP) and that her current diagnosis would be "organizing pneumonia with residual fibrosis." (***Id.*** p. 61, 106-12). The term UIP is now reserved for those people who have progressive decline. (***Id.*** p. 107).

Dr. Wagner stated his understanding that environmental conditions in Kazakhstan were notorious both in the pulmonary community and the nonmedical community, due to a lot of heavy industry, and he first learned of the problem from a friend who did tuberculosis work in Eastern Europe. (***Id.***, p. 17; 90-91). He also reviewed the CIA Fact Sheet on Kazakhstan. (***Id.***, p. 79).

When asked his medical opinion as to the cause of the pulmonary fibrosis which he diagnosed, he testified:

> A. Well, I think that her illness began in Kazakhstan as a consequence of the environmental conditions in Kazakhstan. . . .

(***Id.*** p. 18; ***see also*** p. 140). He indicated that he would have the same opinion whether the condition was UIP or organizing pneumonia with residual fibrosis. (***Id.*** p. 62). However, he could not identify the etiological agent responsible, and he conceded that he would not be able to state with a reasonable degree of medical certainty that UIP was caused by a particular agent. (***Id.*** p. 66, 149-51). If a cause were identified, it would no longer be idiopathic. (***Id.*** p. 67). Although stating his opinion to a reasonable degree of medical certainty, Dr. Wagner appeared to be using the term "reasonable medical certainty" to apply to a situation where there was less than a fifty percent likelihood, however. (***Id.*** p. 174-77). However, he later indicated that he had ruled out other possible etiologic factors. (***Id.*** p. 178-80).

When asked on cross examination whether he had determined another case of UIP was caused by environmental factors, he stated that "we don't know because we don't really know what the, the immunologic insult which leads to the inflammatory process" but that "[u]ltimately, you could say they are all caused by environmental processes, if they, if some things have been excluded, if viral infection has been excluded," and he noted that a group of people have familial pulmonary fibrosis. (***Id.*** p. 62). He determined that Claimant did not have a familial history and he indicated that viral illnesses could be excluded based upon the lung biopsies. (***Id.*** p. 63-65). Dr. Wagner also indicated that, although the disease was idiopathic, meaning that "the cause is not definitely known", there were other factors that put someone at risk for the disease, including smoking (which resulted in a twofold increased risk) and locale (with an increased risk in areas with industry, agriculture, or heavy pollution). (***Id.*** p. 65-66, 68-79). Later he mentioned certain antibiotics (such as nitrofurdation, which has been associated with pleural fibrosis and retroperitoneal fibrosis, but not pulmonary fibrosis). (***Id.*** p. 92-94). Parasites have not been implicated, but "could be"; the only parasite so far associated with lung disease is the lung fluke found in southeast Asia. (***Id.*** p. 94). Dr. Wagner defined a risk factor as "a cause that has not reached the next level of certainty." (***Id.*** p. 67). He agreed that Claimant's 40 pack year smoking history was significant as a risk factor. (***Id.*** p. 70-71). However, he later indicated that smoking was probably not the causative factor, because the condition did not flare up when she was tapered off the steroids and had resumed smoking. (***Id.*** p. 84-85). Age is another risk factor (although not an independent one), and as people become older they are more likely to develop UIP. (***Id.*** p. 101-02). In the absence of collagen vascular diseases, the condition is "actually a bit more common in men." (***Id.*** p. 102-03). Certain antidepressants, known as tricyclics, are also risk factors, and he did not know whether she was exposed to tricyclics; she was taking Prozac, which is not a tricyclic. (***Id.*** p. 103-04). He has not seen any of the other drugs she was on (Voltar, a nonsteroidal antiinflammatory agent; Crimpro, a combination of Premarin and progesterone; Percodan, an oxydodone and aspirin; or Prozac, an SSRI) as being associated with UIP. (***Id.*** p.106).

Dr. Wagner referred to a number of studies, including a study finding an increased incidence in industrial areas as compared with other areas in the United Kingdom, a Japanese study finding an increased incidence in rural areas as compared with other areas, an occupational study finding an increased incidence in hairdressers, and a Mexico City study finding an increased incidence in outdoor shoe shine people (***Id.*** p. 71-78). Taken together, Dr. Wagner opined that these studies suggest that particulates in the air are best correlated with the risk. (***Id.*** p. 78). He was unaware of any studies for Kazakhstan, and he has never been there. (***Id.*** p. 79). Nevertheless, he advised Claimant not to return to Kazakhstan because of the potential risk. (***Id.*** p. 80-85).

When asked whether Claimant developed UIP in February or March 1998, when she first became symptomatic, Dr. Wagner said that he was "guessing that she probably was in the early stages of it" and he would "guess" that her immunologic insult started or

began "in the few weeks to month, maybe six weeks before that time." (***Id.*** p. 96). However, he could not exclude the possibility that she had UIP before she went to Kazakhstan in November of 1997. (***Id.*** p. 97-98). Based on the degree of the total lung involved at the time of the lung biopsy, and the amount of inflammation (cellular component) as opposed to scarring (fibrotic component), he determined that it was most likely that the onset was when she was in Kazakhstan. (***Id.*** p. 97-101).

Dr. Wagner testified that, although they had been able to arrest the progression of her disease and Claimant was "still capable of getting around", she had some permanent impairment. (***Id.*** p. 18). Dr. Wagner opined that that impairment would probably not prevent her from working as a lawyer. (***Id.*** p. 145-46). At the time of the deposition, Claimant's diffusing capacity was down to 50% of normal or slightly less and it would be "forever abnormal." (***Id.,*** p. 26-27). That is not to say that she has lost half of her diffusing capacity, however. (***Id.*** p. 145). While the last bit of data indicated only age-related decrements in diffusing capacity, she is on a curve which parallels the normal curve, but is lower. (***Id.*** p. 28). Diffusing capacity is a measure of the ability of the body to salvage oxygen from the atmosphere and put it in the blood. (***Id.*** p. 27). Dr. Wagner explained that the observation by Dr. Friedman that Claimant's specific diffusing capacity had reached 75 percent of predicted in 1999 was not useful clinically, because that measurement only measures the health of the alveolar units and could be completely normal in someone who had lost a lung; in contrast, overall diffusing capacity would be reduced. (***Id.*** p. 29-32). Dr. Wagner opined that Claimant's reduction in overall diffusing capacity would cause her to be exercise limited for the rest of her life. (***Id.*** p. 30-31). However, in addition to the diffusing capacity reduction and restrictive lung disease, she has obstructive lung disease caused by her smoking, which would make the lung "look normal" on testing due to an increase in forced vital capacity and total lung capacity. (***Id.*** p. 31-32; ***see also*** p. 51-54).

It was Dr. Wagner's opinion that the Prednisone therapy impacted or aggravated the condition of Claimant's right hip because of its impact on bone density and acceleration of symptoms (pain). (***Id.***, p. 24-26, 128-30). The loss of bone density in Claimant's case was reflected by her loss of about a quarter inch of her femoral height. (***Id.***, p. 25, 123-27, 133). In Dr. Wagner's opinion, "her right hip is now not really an elective procedure anymore." (***Id.***, p. 24-26). Contrary to a statement he made in a letter of March 31, 1999, Claimant did not have avascular necrosis. (***Id.*** p. 120-24). Dr. Wagner saw the pathology report but not the x-rays. (***Id.*** p. 122). She did have preexisting arthritis. (***Id.*** p. 125).

Dr. Wagner also opined that the following conditions were caused or exacerbated by Claimant's Prednisone use, in addition to the right hip condition: (1) the left hip condition and probable need for left hip replacement, due to the same mechanism that aggravated the right hip condition (although he was unable to state that the association was more likely than not); (2) Claimant's herpes zoster or shingles, due to the decline in

immunologic surveillance, and chronic pain syndrome and cellulitis, resulting from the herpes zoster; (3) ulnar nerve problems necessitating the March 2000 surgery, most likely related to the herpes zoster (although he conceded that there was a reasonable medical probability that the nerve problems were unrelated); (4) coronary artery disease/cardiac problems in Sarajevo (leading to angioplasty and stent placement in Vienna), resulting from increase in serum cholesterol levels and acceleration of the atherosclerotic process (although there was an equal chance coronary artery problems would have occurred anyway); (5) body fat and muscle composition changes, resulting in an egg-shaped appearance, including "moon faces" (big round faces) and "Buffalo hump" (protuberance between shoulder blades combined with shrinking of the extremities); (6) Claimant's plastic surgery for the "moon face"; and (7) dental problems due to the immunosuppressant effects on oral health. (***Id.*** p. 33-49, 138-39, 151-59, 162-74). He agreed that Claimant was temporarily totally disabled from June 1998 until she returned to work; from March 3 to 21, 2000; from May 12, 2000 to June 8, 2000; and from September 9, 2000 and continuing as a result of these conditions. (***Id.*** p. 27). He also opined that in the future Claimant could develop other long term effects of Prednisone use, including osteoporosis, hair loss, and cataracts. (***Id.*** p. 50).

Medical Opinion of Carl B. Friedman, M.D.

Carl B. Friedman, M.D., a board-certified internist, reviewed Claimant's medical records on behalf of the Employer and acted as Employer's expert witness. (EX 1; EX 2). He prepared a report dated July 30, 2000. (EX 1). Dr. Friedman's de bene esse deposition was taken on October 25, 2000. (CX K).

In Dr. Friedman's report of July 30, 2000, inter alia, he opined that the Claimant's diagnosis was usual interstitial pneumonitis or idiopathic pulmonary fibrosis; that risk factors for the disease included viral infections, environmental factors (such as exposure to metallic, wood, or inorganic dusts or organic solvents in an indoor environment), cigarette smoking, antidepressants, genetic factors, immunological factors associated with autoimmune deficiencies, myasthenia gravis, celiac disease, and chronic active hepatitis; that "[a]t the present time there is no specific cause and effect relationship that can be ascribed to the development of idiopathic pulmonary fibrosis"; and that "[i]t would be unlikely for [Claimant] to have an industrial exposure that would cause an increased risk for development of usual interstitial fibrosis or IPF in her position as a legal specialist." (EX 1; EX 2). He indicated that notwithstanding the risk factors, for "a large percentage with pulmonary fibrosis, the etiology is unknown." ***Id.***

At his deposition, Dr. Friedman testified that the diagnosis of usual interstitial pneumonitis (or idiopathic pulmonary fibrosis) was nonspecific. (CX K at 8, 40). Dr. Friedman further testified:

> . . . . . In this case, the cause is not defined by the histology. We can't define what happened. So the cause has got to be defined by the rapidity of the onset, her exposures, whether it was industrial exposures, environmental exposure, a drug exposure, a trauma, an inhalation exposure, or an infectious disease exposure. All of those modalities could be responsible for her to have this change in her X-ray.
>
> And even above that, immunological changes within the body, such as in sarcoidosis and rheumatoid arthritis and lupus, these changes occur without any external environmental contributions. You just develop this as an overall disease process.

Id. at 8-9. Dr. Friedman indicated that unless these conditions are treated early, there could be a fixed pulmonary fibrosis that never goes away. ***Id.***

Dr. Friedman testified that possible causes for Claimant's pulmonary condition were antidepressants, antibiotics (such as nitrofuradantin, sulfur medication, and salicylamide) and antimetabolites. ***Id.*** at 18-19. He further testified that cigarette smoking did not cause the condition, but it was a risk factor tha[t] increased her risk by a factor of 2. ***Id.*** at 17.

Dr. Friedman noted the following that he found to be of significance in Claimant's case:

(1) Even though Claimant was a cigarette smoker, she did not come down with acute bronchitis, which would be expected if she had been exposed to a heavily polluted area. From that, he concluded that an industrial exposure was not responsible. ***Id.*** at 13-14.

(2) Based upon the history provided, Claimant's fibrosis did **no**t develop slowly (over a period of six or seven months) but developed over a period of weeks. ***Id.*** at 14.

(3) Claimant was exercise limited or she would have discovered the condition earlier. ***Id.*** at 14-15.

Dr. Friedman stated his opinion that Claimant's pulmonary fibrosis was not caused by inhalation of pollutants or internal hazards in Kazak[h]stan because "histologically, no etiological relationship could be made" and "[i]t's idiopathic why this happened." ***Id.*** at 13-14. Later, he explained:

> Q. Now, you say you don't or most likely were not going to know the cause. Are there things that we can factor out that we've already factored out as non causes?

> A. In my opinion, with reasonable medical certainty, since she didn't have an industrial exposure in the city, that this will not induce pulmonary fibrosis, after a six-month exposure to whatever she was exposed to.
>
> Now, we do know, we do know epidemiologically that there are a higher percentage of people who develop this disease that has a risk factor of metallic fumes, of industrial fumes, but his occurs in people who have an industrial exposure, not a casual street or home exposure to the city as a whole.

Id. at 20. When further questioned about the impact of Claimant's environmental exposure, he went on to say: ". . . I rule it out because she didn't have an industrial exposure." ***Id.*** at 21; ***see also*** 40-47. On cross examination, he further stated that he could rule out the exposure because it was not an occupational exposure, which would involve a long period of exposure and high concentration of exposure. ***Id.*** at 76-77. He could **not** rule out a viral cause. ***Id.*** at 78. He also indicated that he would want a more complete history, including Claimant's drug use and type of exposure. ***Id.*** at 77.

Dr. Friedman also opined that the Claimant's right hip problems, leading to a right hip replacement, were unrelated to the Prednisone as they predated her assignment in Kazakhstan and were caused by osteoarthritis, not avascular necrosis. ***Id.*** at 21-25, ***see also*** 53-61. He similarly concluded that the left hip problems were unrelated, but was unable to rule out the association. ***Id.*** at 25-26, 71-72. Dr. Friedman also opined that the ulnar nerve problem was more likely caused by trauma, not herpes zoster infection, although the infection itself was related to Prednisone treatment, as steroids can enhance the development of shingles. ***Id.*** at 26-30, 65-67, 70-71.

On cross examination, Dr. Friedman admitted that he never met or examined the Claimant and that he was certified in internal medicine but not pulmonary diseases. ***Id.*** at 33. He also admitted that Claimant complained of exercise tolerance problems in February or March of 1998 and that such problems could have been related to the fibrosis. ***Id.*** at 35-38. He further indicated that Prednisone could cause body contour changes (including moon faces and buffalo hump); lipid abnormalities (including elevated cholesterol); imbalance in calcium, nitrogen and potassium and salt retention; fragile skin, bruising, and stretch marks; osteoporosis and osteonecrosis of bone ends; cardiovascular changes and congestive heart failure; psychoses; and infectious and immune changes (including increased susceptibility to infections and suppression of immune response.) ***Id.*** at 62-66. He also agreed that the high dose steroids could have elevated Claimant's cholesterol and possibly aggravated her coronary problems (obstructive coronary artery disease), but noted that smoking was another risk factor. ***Id.*** at 72-74.

Miscellaneous

As discussed above, I have admitted into evidence Claimant's Exhibit D ("CX D") (appearing in Binder A, pages 34 to 99), consisting of newspaper articles and research documents from the C.I.A. and United States Energy Information Administration sites on the world wide web. However, I found that Employer's objections (primarily, that the records relate to other parts of the former Soviet Union and not to Almaty, Kazakhstan, where Claimant was employed) would be considered in weighing the evidence. The evidence consists of:

(1) an undated newspaper article relating to high levels of pollutants (industrial wastes); a finding of lead, arsenic and cadmium poisoning in two thirds of the children; and high levels of congenital defects, central nervous system disorders, cancer, and other major diseases in Karabash, Russia, located in the foothills of the Ural Mountains, near Russia's southern border with Kazakhstan.

(2) an undated C.I.A. online Factbooks for Kazakhstan, which states:

> **Environmental–current issues:** radioactive or toxic chemical sites associated with its former defense industries and test ranges are found throughout the country and pose health risks for humans and animals; industrial pollution is severe in some cities; because the two main rivers which flowed into the Aral Sea have been diverted for irrigation, it is drying up and leaving behind a harmful layer of chemical pesticides and natural salts; these substances are then picked up by the wind and blown into noxious dust storms; pollution in the Caspian Sea; soil pollution from overuse of agricultural chemicals and salinization from faulty irrigation practices.

Almaty is shown near the border of Kyrgyzstan in the lower right hand (Southeast) corner of the map of this vast country of Kazakhstan, the second largest of the former Soviet republics, encompassing 2,717,300 square kilometers.

(3) December 1998 United States Energy Information Administration fact sheets on Azerbaijan (on the Caspian Sea, across from Kazakhstan) and for the whole Caspian Sea Region (which lies along Kazakhstan's western border, to the south), reflecting energy-related difficulties in that region.

(4) articles dated in December 1993, September 1992, September 1993, and November 1995 relating to radiation pollution in the former Soviet Union.

(5) an undated C.I.A. online Factbook for Kyrgyzstan, which lies to the south of Kazakhstan reflecting water pollution, water-borne diseases, and soil salinity from faulty irrigation practices in this largely agricultural republic of the former Soviet Union, and an

undated "State of the Environment of Kyrgyzstan" report indicating pollution from pesticides, poisonous industrial waste, food wastes, and radioactive pollution.

Summary of Evidence and Facts in Current Proceedings

The evidence before me at the most recent hearing consists of the testimony of Ms. Cohen and Dr. Wagner, the deposition of Dr. Friedman, and various articles and other records.

Claimant's July 22, 2002 Testimony

As before, Claimant was a credible witness. She reiterated her history of employment in Kazakhstan from November 1, 1997 through May 31, 1998, the bulk of which occurred in Almaty, and her subsequent hospitalization at George Washington University Hospital shortly after she returned to the United States, on June 6, 1998. (TR2 at 23-24). She further reiterated her testimony as to the black cloud – which she described as "huge blackness and clouds in front of [her]" – when she approached Almaty on her way from Kyrgyzstan. (TR2 at 24). She indicated that she had not reflected much upon the atmosphere in Almaty while she was employed there, but that in retrospect she recalled that the ground was covered with a six- to eight-inch-thick layer of black ice, which remained frozen under a sunless, gray sky. (TR 2 at 25 to 26). She also was unable to see the mountains surrounding the city from the downtown due to the atmosphere. (TR2 at 59-60). Using maps of Almaty (CX L1, L33) for illustrative purposes, she testified that she lived and worked in zones in the city which were identified as the most polluted, according to an article on the Internet prepared by an organization known as the Greenwomen (CX L1).[20] (TR2 at 28 to 31, 52 to 53). Her office was six to eight blocks from where she lived, but she had a driver who took her to work. (TR2 at 53 to 54). Approximately 90% of her time was spent at her home or the office, while the rest of her time was spent at restaurants or shopping, and she also went to Banking Commission meetings. (TR2 at 54-55). She frequently observed trash being burned in the city of Almaty, in apartment buildings right around the corner from her. (TR2 at 31). In May of 1998, she also visited another site downtown, an investment house at a factory-type converted building where she taught the Securities Commission staff how to do inspections. (TR2 at 54). At that time, she was experiencing the diarrhea symptoms and the shortness of breath. (TR2 at 54). The breathing problems dated from April or May of 1998. (TR2 at 58). She does not recall having had a metal taste in her mouth, chronic flu-like symptoms, or chronic coughing. (TR2 at 58-59). After she returned from Latvia, in April of 1999, she began researching the issue of Kazakhstan's environment, but it was difficult for her to obtain information, a matter which she attributed to the repressive nature of the society based upon recent press accounts. (TR2 at 31 to 33, 36 to 38). Claimant read the Greenwomen article about Almaty, which described its location amidst the mountains, with little air flow, strong temperature inversions, and frequent fog, resulting in the accumulation of harmful substances, and she noted that these comments were consistent with her personal observations. (TR2 at 38 to

[20] The evidentiary value of this and similar articles will be discussed ***infra.***

41). However, she did not notice whether there were any plants or factories in the neighborhoods where she lived and worked. (Tr. 56).

On cross examination, Claimant indicated that she had missed more time since the previous hearing and had been out of work due to left hip replacement surgery from October 27, 2000 until June 1, 2001, and she claimed entitlement to temporary total disability benefits for that period. (TR2 at 42). Claimant further indicated that she had been working "off and on" since June 1, 2001. (TR2 at 42-43). She was also claiming temporary total disability benefits from June 6, 1998 to February 1, 1999 (for pulmonary fibrosis, partial lung biopsy); from March 3, 2000 to March 21, 2000 (for left ulnar nerve surgery), and from May 14, 2000 to June 8, 2000 (for carbon stint implant in the heart). (TR2 at 43-44). Since the prior hearing, in addition to the left hip replacement surgery, she had "enormous dental surgeries," which she paid for entirely. (TR2 at 48). GW Health Insurance paid for the left hip surgery, but she paid for the nursing and drugs. (TR2 at 48). Since the prior hearing, she has also paid for ongoing doctor visits and prescriptions related to her claim. (TR2 at 48). Claimant has not seen Dr. Wagner since the last hearing because he is not in practice at GW any more, and she has not seen her current pulmonologist, Dr. Morgan Dulaney, for at least one year. (TR 2 at 48-50). Claimant is also seeing a cardiologist, Dr. George Bren, but she feels that she is doing well because of the Lipitor she is taking (TR2 at 50). Her last contract overseas was from June through October of 2001 in Montenegro, where she worked as a securities advisor, but she does not plan to continue with that kind of work. (TR2 at 50-51).

Dr. Randall P. Wagner's July 22, 2002 Testimony

Dr. Wagner was a highly credible, persuasive witness. As noted above, Dr. Wagner provided his ***de bene esse*** deposition in connection with the previous proceedings. At the time of the second hearing, he was employed by Mid-Atlantic Critical Care doing inpatient critical care medicine, and he was no longer the Claimant's treating physician, having given up his standard pulmonary practice. (TR2 at 61-65, 117-118). Dr. Wagner has impressive credentials. As at the time of the previous hearing, he is still board certified in internal medicine as well as the subspecialties of pulmonary diseases and critical care medicine.[21] (CX J; TR2 at 62). Dr. Wagner has also had considerable teaching experience, particularly at George Washington University, and he continues to teach at George Washington Hospital and the Adventist Hospital. (TR2 at 63). He has also been involved in clinical and scientific research, and his current protocols include one studying the prevention of ventilator-associated pneumonia. (TR2 at 64). Basically, he describes his role as that of "the ICU pulmonologist", which means that he handles the critical care pulmonary work in his practice. (TR2 at 64-65).

[21] Dr. Wagner's credentials were discussed at his ***de bene esse*** deposition conducted on October 26, 2000 in connection with the previous proceedings, and his outdated curriculum vitae appears as Exhibit 1 to that deposition. (***See*** CX J).

Dr. Wagner rehashed some of the matters relating to Claimant's treatment that he discussed at his ***de bene esse*** deposition of October 26, 2000 (CX J [transcript of deposition]). He first saw Claimant on June 8, 1998, at which time she was in the intensive care unit, intubated (with an endotracheal tube supplying oxygen) and on mechanical ventilation. (TR2 at 65). When she was conscious enough to provide a history, Claimant recounted the shortness of breath and exercise limitations she had suffered in Kazakhstan, dating from March, and he also obtained a history from her chart and from her daughters. (TR2 at 65 to 66, 74, 129). Although the impression of the physicians who had initially seen her was that she had bacterial pneumonia, she had deteriorated after two or three days of "good antibiotics," which suggested to Dr. Wagner that either the wrong antibiotics were being used or the condition was not sensitive to antibiotics. (TR2 at 69). Dr. Wagner performed a bronchoscope, which consisted of two tests (a broncho alveolar lavage and removal of transbronchial tissue), followed the next day by an open lung biopsy. (TR2 at 68-71). Based upon the pathology report, Dr. Wagner concluded that there was no evidence of a viral etiology because "[t]here were no inclusion bodies, there were no granules, there just wasn't anything to suggest in either the alveolar lining cells or the lipocytes or in the macrophages that there was a virus there." (TR2 at 69-70). The biopsy revealed the spectrum of an ongoing process, consisting of relatively normal tissue, acute and active inflammation in the breathing units and interstitial area, loose connective tissue (fibrin and collagen) in the interstitium and alveolar space, mature collagen that had not yet reached the scar level in the peripheries of the lung, and places that were fibrotic and no longer involved in the active inflammatory process. (TR2 at 70-74). In view of the absence of honeycombing of the lungs on a high resolution CT scan, the mature scarring reflected an insult that "probably occurred three, maybe at most, four months before that," placing the onset in approximately February or the beginning of March, 1998. (TR2 at 74 to 75, 114). An onset at that point would be consistent with Claimant's symptoms of shortness of breath and exercise intolerance, which are reflective of pulmonary fibrosis, as well as with her clinical course. (TR2 at 75-76, 115). Dr. Wagner disagreed with Dr. Friedman as to the rapidity of the onset of the disease, the significance of whether she had symptoms of bronchitis at the time of the onset, and the possibility of a viral etiology. (TR2 at 77 to 80). Dr. Wagner ruled out a viral etiology based upon the pathology reports and specifically the findings on broncho alveolar lavage – a matter which is of significance as certain viral infections would have mandated different treatment. (TR2 at 81 to 83). Based upon the histologic diagnosis of UIP [usual interstitial pneumonitis], he treated Claimant with very high dose steroids. (TR2 at 83 to 85). Initially, he determined that Claimant had idiopathic pulmonary fibrosis, which meant that he did not know the cause. (TR2 at 86).[22]

Prior to testifying at his deposition in the previous case, Dr. Wagner knew from colleagues and friends that Kazakhstan was not a pleasant place from a pulmonary standpoint and the air was

[22] Dr. Wagner explained that a diagnosis of usual interstitial pneumonitis (UIP) was a tissue diagnosis, based upon a pattern of inflammation and fibrosis on tissue. When that pattern is combined with a consistent type of course and the lack of inciting factor, the term idiopathic [mistranscribed as "etiopathic"] pulmonary fibrosis is used. (TR2 at 87). For ease of reference, the term "pulmonary fibrosis" will be used herein to describe Claimant's pulmonary condition and is intended to be inclusive.

dirty.[23] (TR2 at 87-88). Since the date of his previous deposition, he has learned more about the environment there based upon sources that are not in the medical literature, where he would typically look. (TR2 at 88 to 89). Specifically, he reviewed the evidence in binder L [CX L1 through CX L33]. (TR2 at 88-101, 115). These sources included the United Nations Agenda 21 article, which reported that in 1995, one out of every four persons in Kazakhstan suffered a respiratory disease, with the greatest incidence in Almaty and three other areas. (TR2 at 92-93, CXL6). He also reviewed the Greenwomen's article, which discussed a longitudinal observational study conducted by the Ministry of Health, relating to children located in three zones within the city of Almaty; the study ostensibly showed a higher rate of respiratory disease and death in the areas Claimant lived and worked. (TR2 at 91-92, CX L1). Dr. Wagner explained his understanding that cadmium was a pollutant in the atmosphere of Almaty, resulting from three sources outside of the city (the Lake Balkhash area, the mines and smelting operations at Shymken [mistranscribed as Skimaten], and the Aral Sea itself and its dried seabed) and three sources within the city (coal use, a small-arms ammunition plant, and trash burning). (TR2 at 98-99, 102-113, 204-205). Based upon this additional information, Dr. Wagner opined that the Claimant's exposure to cadmium in the Almaty atmosphere caused Claimant's UIP. (TR2 at 115-116). The clinical diagnosis would be Cadmium lung. (TR2 at 119).

On cross examination, Dr. Wagner indicated that at the time he treated Claimant, he had ruled out viral and bacterial as the cause of Claimant's UIP, but he had not looked at cadmium or any toxic metals as a cause. (TR2 at 121). A bacterial etiology was ruled out based upon the Claimant's clinical findings, including blood cultures, sputum cultures, broncho alveolar lavage fluid cultures, and lung biopsy cultures (including a Warthin Starry stain). (TR2 at 122-125). At that point in time, in June 1998, he thought that he was looking at an immunologically mediated event, meaning that the etiology was unknown but that the immune system had been activated, which could also be indicative of exposures to drugs (such as ergot alkaloids used to treat Parkinson's and migraine headaches, various drugs used to treat cardiac rhythm disturbances, and Macrolide used to treat urinary tract infections) and environmental exposures.[24] (TR2 at 126-127). At that time, he considered toxic exposure as being the cause, and he testified at a deposition about agents in the dirty air being responsible. (TR2 at 130). At the hearing before me, he testified that he is now "quite sure" that cadmium was the causative agent, based upon information he has obtained since the last hearing, and specifically that provided by Dr. Friedman. (TR2 at 131 to 134). When given a definition of reasonable medical certainty as meaning a probability of greater than 50 percent, he stated that the criteria had been satisfied. (TR2 at 200-204). He explained that he continued to hold the opinion that dirty air, or toxic exposure, was the

[23] Dr. Wagner testified that he has friends who visited Kazakhstan and worked with either the World Health Organization ofrthe Work Bank "doing health studies, mostly tuberculosis stuff." (TR2 at 90-91).

[24] On questioning from the undersigned, Dr. Wagner indicated that the antibiotics that Claimant had received for urinary tract infections (such as Peridium) were not associated with pulmonary fibrosis. (CX2 at 206-208).

cause, as he had at the time of the earlier proceedings, but he now was able to "put a name to the toxic exposure." (TR2 at 202). He conceded that he had never diagnosed a cadmium lung disease before, nor has he ever ordered a cadmium test. (TR2 at 203).

Dr. Wagner was cross examined concerning the peer reviewed articles concerning cadmium exposure relied upon by Dr. Friedman.[25] (TR2 at 134-140, 173, 180-188, 190-198; EX E2). He agreed that the Greenwomen article (CX L1) was not peer reviewed and he testified that attempts to obtain the cited data from the Ministry of Health were unsuccessful, so he relied upon the Greenwomen's recounting of the data. (TR2 at 141-142, 155-158). He also indicated that he had relied upon some of the other articles from Internet and other non-peer-reviewed sources (specifically CX L3, L6, L7,L10, L11, L15 and L16) to support his conclusion that Claimant was exposed to cadmium. (TR2 at 143-155, 158-173). He also discussed the mechanisms by which cadmium can be absorbed by the body, including ingestion (drinking) and inhalation, and his opinion is that Claimant got her cadmium exposure by inhalation. (TR2 at 174-175). Acute exposures may be characterized by adult respiratory distress syndrome. (TR2 at 175-176). Symptoms of acute catastrophic exposures would be headaches early on followed by coughing later, and there could also be a metallic taste, diarrhea, and possible vomiting. (TR2 at 175-177). Dr. Wagner opined that Claimant's exposure was acute but not catastrophic, and was probably characterized by several non-catastrophic, recurrent exposures over three or four months, and continuous low-level exposure, which was sufficient to cause her disease. (TR2 at 178-179, 189, 202-203). He conceded that Claimant's situation did not exactly fit the pattern of either single acute exposures or long-term protracted exposures that were the subject of the articles attached to Dr. Friedman's deposition. (TR2 at 197 to 198).

The undersigned questioned Dr. Wagner about other etiologic factors. He indicated that smoking had not been shown by the studies cited by Dr. Friedman to be a causative agent, although others found smoking to be a risk factor, and he explained why he had essentially ruled out other possible causes, including collagen vascular disease (for which she was worked up) and drug toxicities. (TR2 at 206-214). If it were of viral etiology, the latent period would depend upon the virus and could be as little as four or five days or as much as years, or even decades. (TR2 at 218-219). He also explained that the term usual interstitial pneumonitis is merely a pattern of the tissue, which can exist regardless of the causative agent, but there has to be an appropriate clinical course for the diagnosis of idiopathic pulmonary fibrosis to be made. (TR2 at 213). Once a causative agent is found for the fibrosis, it is no longer "idiopathic." (TR2 at 211). Typically, patients with idiopathic pulmonary fibrosis do not survive for four years, as Claimant has. (TR2 at 212).

On recross, Dr. Wagner testified that the fixed fibrosis (or scarring) of cadmium lung exposure would not be resolved with steroids, but that the steroid therapy was effective on the inflammatory part of the process in the case of a long-term chronic exposure. (TR2 at 214-215).

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[25] On redirect, he explained that the Perry article showed that every individual reacts differently to cadmium. (TR2 at 205-206).

For an acute exposure, such as inhalation of a noxious gas, the persons exposed will become "very, very sick early on, but when they heal, they don't heal with scar, they regenerate." (TR2 at 216-217). In such a case, steroids would do no good. (TR2 at 216-217).

Dr. Carl Friedman's July 10, 2002 Deposition

Dr. Friedman's ***de bene esse*** deposition was taken on July 10, 2002 in Cedarhurst, New York, and both parties participated. The deposition transcript was admitted into evidence as Employer's Exhibit E2. Dr. Friedman's April 15, 2002 report appears at Employer's Exhibit E1. At the previous hearing, Dr. Friedman's curriculum vitae was admitted as Employer's Exhibit 2. (TR1 at 84 to 85).

At his most recent deposition, Dr. Friedman, who is board certified in internal medicine, testified that he is still in practice on Long Island, treating primarily pulmonary patients. (EX E2 at 3-4.) His patients include those who suffer from occupational pneumoconiosis, silicosis, asbestosis, inhalation injuries, asthma, pneumonia, tuberculosis, and lung cancer, as well as those who suffer from hypertension. ***Id.*** His hospital affiliations have not changed since the prior hearing and he has continued to testify in worker's compensation cases; he has also recently attended a conference at Mt. Sinai on interstitial lung disease, including usual interstitial pneumonia (UIP), and its treatment. (***Id.*** at 4-5). In addition to the traditional treatment of UIP with steroids, which did not work in some of the cases, there is a new treatment that focuses on the fibrogenic focci (which create fibroblasts), which are responsive to interferon alpha. (***Id.*** at 6-7). Dr. Friedman stated that it is necessary to do a lung biopsy to eliminate other common diseases like sarcoidosis or bronchiolitis obliterans with organizing pneumonia, which respond to steroids, as opposed to fibrosis due to chronic cadmium exposure, which does not respond to anything. (***Id.*** at 6-7). Since the previous trial, Dr. Friedman had reviewed various records submitted by or relating to the Claimant. (***Id.*** at 7-8, 25-26). However, he has never examined her nor has he spoken with Dr. Wagner or any of her other treating physicians. (***Id.*** at 25, 56-57). His review was confined to the medical records and he did not review x-rays. (***Id.*** at 62).

Dr. Friedman explained that there were two types of illnesses one can get from cadmium: (1) pulmonary fibrosis resulting from long-term exposure, as in the case of low level occupational exposure over a period of years and (2) acute pulmonary edema resulting from short-term exposure to high levels of cadmium, which occurs within seven days from exposure and results in residual fibrosis (which may improve) if the individual recovers. (***Id.*** at 8-11, 13). In the case of acute pulmonary edema, the kidneys are affected first, due to low molecular weight protein urea, followed by the lungs. (***Id.*** at 9-10). Within minutes of an acute exposure, it will cause coughing, cyanosis (turning blue), and shortness of breath. (***Id.*** at 14-15). For a chronic condition, coughing and shortness of breath would appear first, and then pulmonary function tests would show an abnormal restricted component in the breathing. (***Id.*** at 14). Dr. Friedman worked for a jewelry company that did soldering, with solder found to be contaminated with cadmium that had been used as a flux, and over the years some of the older workers developed pulmonary fibrosis "very minimally." (***Id.***). Dr. Friedman concluded that if Claimant's condition were attributable to

cadmium, it would have to be an acute exposure to a high concentration at an industrial level, and such a condition would not be responsive to steroids. (***Id.***). In the occupational setting of solderers in a factory who do not have high levels of exposure, it would take a minimum of three to four years for fibrosis to develop, but he does not know how long it would take in a nonindustrial setting. (***Id.*** at 13-14). There are no papers in the literature that Dr. Friedman is aware of which would support the position that less than a six-month exposure to cadmium in an environmental nonindustrial setting can cause pulmonary fibrosis, because chronic exposure at low levels takes years for the development of pulmonary fibrosis. (***Id.*** at 13, 73).

Dr. Friedman also testified that exposure to arsenic, chrome, and lead from leaded gasoline would not cause pulmonary fibrosis, and that hydrocarbons usually do not cause it. (***Id.*** at 10-11). Sulfuric acid is unlikely to cause fibrosis but when it does so, the fibrosis is accompanied by upper respiratory tract symptoms including coughing. (***Id.*** at 11-13).

Dr. Friedman discussed articles in various journals which discussed occupational cadmium exposures, copies of which were attached to the deposition transcript, including (1) Exhibit A, a review article entitled "Acute Occupational Cadmium Poisoning" from the January 1967 Journal of Occupational Medicine, a peer-reviewed journal [Dunphy]; (2) Exhibit B, a case history entitled "Abnormalities in pulmonary function after brief exposure to toxic metal fumes" [CMA Journal, September 23, 1978, Anthony, Zamel, Aberman]; (3) Exhibit C, "Lung function in workers using cadmium containing solders" [British Journal of Industrial Medicine, 1986, Edling, Elinder, Randma]; (4) Exhibit D, "Mortality of cadmium-exposed workers, A five-year update" [from Scan J Work Environ Health (1988),Kazantzis, Lam, Sullivan]; (5) Exhibit E, "Pulmonary Effects of Chronic Exposure to Airborne Cadmium" [American Review of Respiratory Disease, 1976, Smith, Petty, Reading, Lakshminarayan]; and (6) Exhibit F, "Cadmium-Induced Lung Injury: Chronic Cell Kinetics and Long-Term Effects" [Toxicology and Applied Pharmacology, 1985, Martin, Witschi]. (***Id.*** at 15-21, 51-55; ***see also*** EX E2 Exhibits A through F). Dr. Friedman conceded that the Smith study (Exhibit E) showed that the amount of cadmium inhaled or ingested by an individual will affect each individual differently. (***Id.*** at 51-55).

Based upon his review of the literature and the additional documents provided by Claimant, Dr. Friedman stated that he had not changed his opinion since the prior trial, which was that Claimant had developed acute respiratory insufficiency with "evidence of bilateral pulmonary infiltrates with biopsy that reflected usual interstitial pneumonitis" and that it was not due to her exposure to urban pollution but was most likely triggered by a virus, and that she was made susceptible to development of the condition by her cigarette smoking (EX E2 at 21-22, 40). A virus results when there is viral pneumonia and "a triggering of fibrotic response and the way the lung reacts is uniformly in one direction to an irritant or infection." (***Id.*** at 23). A virus may have been the cause of the diarrhea that Claimant experienced, or it could have been due to some other etiology, such as a parasitic infection. (***Id.*** at 23-24, 71-72). The onset of the Claimant's condition could have been days from contracting the virus. (***Id.*** at 24). In this regard, Dr. Friedman had a patient who presented with a cough and viral pneumonia and came back ten days later with bronchiolitis obliterans, which was a response to the bronchial infection. (***Id.***). Another

such response to infection could be usual interstitial pneumonitis. (***Id.***). It is still Dr. Friedman's opinion that a viral infection was probably the cause, and she may have contracted it in Kazakhstan. (***Id.*** at 69-71).

On cross examination, Dr. Friedman was asked about the Greenwomen's report, which he criticized as being produced by an "advocate group of people" and "not peer-reviewed." (***Id.*** at 28-31, 34-35, 78-80). Later, he conceded that he had not read it, nor had he read the other articles attached to Claimant's motion for modification, although they were apparently provided to him. (***Id.*** at 60-61, 78). He did, however, read the articles submitted during the previous proceedings. (***Id.*** at 72-75). He stated that there was no medical evidence to show that "people who are in Almaty and exposed to cadmium at the dose level have an increased rate of developing a pulmonary fibrosis," and he also stated that there was no scientific evidence that Claimant was at an increased risk of developing UIP because she stayed in the city. (***Id.*** at 31). When asked about the reported higher incidence of mortality in the area of Almaty with higher pollution levels, he indicated that people who have an obstructive lung disease do poorly when exposed to pollution and may develop pneumonia due to increased secretions. (***Id.*** at 33-34). The statement in his report that exposure to metallic dusts, inorganic dust, organic solvents or wood or metal dusts in an indoor environment are risk factors for the development of UIP was based upon his review of the literature relating to wooden stoves and Eskimos. (***Id.*** at 39, 40-41). Dr. Friedman readily admitted that he did not know what levels of cadmium were present in the atmosphere of Almaty, although he suggested that an inference can be drawn that there are not high levels of cadmium from the fact that half of the population does not have pulmonary fibrosis. (***Id.*** at 77). On redirect, he explained that one would expect high levels in an indoor setting as opposed to an outdoor one. (***Id.*** at 81).

Dr. Friedman was questioned about the Claimant's medical records, and he conceded that statements from his report were based upon what he read in the records. (***Id.*** at 36-39, 43-47). He stood by the statement in his report that there was progressive improvement in her serial pulmonary function tests because "[t]he only thing that didn't really improve as dramatically" was the DLCO (diffusing capacity for carbon monoxide). (***Id.*** at 37-38, 41-43). In his opinion, 90 percent of Claimant's condition has resolved and he would expect her to improve further. (***Id.*** at 43, 67-68). However, he explained on redirect that mature scarring or fibrosis (as opposed to inflammation) does not resolve. (***Id.*** at 81-82). Dr. Friedman continues to believe that the Claimant's long history of cigarette smoking aggravated her condition, because smoking causes some fibrosis and is another irritant exposure that can cause bronchitis and changes (small interstitial opacities) in the interstitium of the lung. (***Id.*** at 47-49). When asked about the symptoms Claimant experienced in Kazakhstan, Dr. Friedman testified that shortness of breath was a symptom of interstitial fibrosis but that diarrhea was not, although it was a symptom of cadmium poisoning. (***Id.*** at 49-51). As he stated in his report, in addition to diarrhea, symptoms of acute cadmium exposure include nausea, vomiting and hyperpyrexia. (***Id.*** at 76; ***see also*** EX E1). He went on to state that whether Claimant's respiratory symptoms included bronchitis was not of particular significance as to cadmium exposure because bronchitis can be associated with

pollution of any sort. (EX E2 at 75)[26]. When asked about the biopsy findings of mature fibrosis, immature fibrosis, and active inflammation six days after the Claimant's return to the United States, Dr. Friedman testified that he had been unaware of those findings and only knew about the diagnosis of usual interstitial fibrosis. (***Id.*** at 51, 65-66). However, he testified that the biopsy findings are nonspecific unless sarcoidosis is involved. (***Id.*** at 67-78). He indicated that very rapid progression of scarring occurs with UIP, which starts with pulmonary edema which resolves and then recurs, followed by fibroblastic proliferation. (***Id.*** at 66-67). However, mature scarring would take longer than six days. (***Id.***).

Dr. Friedman was also asked about the side effects of high-dose prednisone, as he had been at the previous hearing, and he reiterated that it could cause elevated cholesterol and was a risk factor for developing coronary artery disease. (***Id.*** at 63-64).

Exhibits from Second Hearing and Evidentiary Issues

Claimant's exhibits from the second hearing consist of a collection of articles obtained primarily from the Internet that appear in Binder L and are designated CX L1 (the Greenwomen article), CX L3 through L8, and CX L10 through L31. A large map of Kazakhstan is designated as CX L32 and a map of the city of Almaty is CX L33. CX L2 is an April 5, 2001 affidavit from Claimant relating to where she worked as compared to the discussion of the epidemiological study of Almaty in the Greenwomen article and CX L9 is Dr. Wagner's September 12, 2001 affidavit; both were submitted in support of Claimant's September 19, 2001 modification request (along with various articles). At the behest of the undersigned (***see*** TR2 at 220-221), Claimant submitted along with her brief the Affidavit of Elaine Shepard (a research assistant to Claimant's counsel) dated September 23, 2002, which I have marked as ALJ 1 and have admitted into evidence herein. In that Affidavit, Ms. Shepard describes how she located the bulk of the articles appearing in Binder L. Claimant's Exhibits CX L1 through L33 were admitted over Employer's objections on the "grounds of hearsay, irrelevancy and prejudice," which I determined would go to the weight of the evidence.[27] (TR2 at 219-220). CX L34, an Internet version of a June 10, 2002 article from the Washington Post, was also conditionally admitted over Employer's objections based upon lateness, lack of relevance, and lack of foundation, and I determined that absent a motion to strike, I would take the objections into account in weighing the evidence. (TR2 at 33 to 36). As I did in the previous hearing, I found that the exhibits would be admissible to the extent that they formed a basis for the expert witnesses' opinions if for no other purpose. (TR2 at 27).

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[26] Dr. Friedman backed off somewhat from his previous deposition testimony indicating that the absence of bronchitis and rapid development of symptoms negated exposure to pollutants as a cause of Claimant's pulmonary fibrosis. (EX E2 at 76-77).

[27] Although the Employer claimed prejudice, none was shown and no basis for the assertion was provided, either at the hearing or in post-trial briefing. (TR2 at 219-220).

Employer's exhibits for the second hearing consist of the April 15, 2002 report of Carl B. Friedman, M.D. (EX E1) and Dr. Friedman's ***de bene esse*** deposition (together with Exhibits A through F, articles from medical journals) (EX E2).

In considering the above exhibits, I have taken into consideration the objections raised by the Employer to them. Specifically, the Employer argues that the articles submitted by Claimant are "unreliable, presenting statements of hearsay and double hearsay, and lacking in any foundation." (Post Trial Brief of Employer and Carrier ["Employer's Brief"] at p. 5, 10-11). Relying on ***Daubert v. Merrell Dow Pharmaceuticals, Inc.,*** 509 U.S. 579 (1993), which charges trial judges with the responsibility of being "gatekeepers in excluding from evidence unsupported expert theories," Employer argues that "Dr. Wagner's unsupported, unreliable, and unverified theory of causation should not be considered by the court." (Employer's Brief at 35). However, with limited exceptions (as set forth in the regulations), common law or statutory rules of evidence and technical or formal rules of procedure are not applicable to cases brought under the Longshore Act and its extensions, and the Benefits Review Board has specifically held that ***Daubert*** does not apply to Longshore cases. ***See*** 33 U.S.C. § 923(a); 20 C.F.R. § 702.339; ***Jones v. Aluminum Company of America,*** 35 BRBS 2001, 2001 WL 467885 n. 4 (BRB April 9, 2002), ***citing Casey v. Georgetown University Med. Center,*** 31 BRBS 147, 152 (BRB Oct. 28, 1997). Nevertheless, the Act and regulations charge me with the responsibility of making investigation or inquiry and conducting the hearing in a manner that will best ascertain the rights of the parties. 33 U.S.C. § 923(a); 20 C.F.R. § 702.339. Thus, evidence which falls short of satisfying the standards for relevance and reliability under ***Daubert*** may lack sufficient probative value to be deemed substantial evidence. ***See O'Kelly v. Department of Army,*** 34 BRBS 39 (BRB 2000), ***on remand,*** 35 BRBS 203 (2001) (ALJ).

After having considered the arguments raised by both parties, I assign little weight to the articles appearing as CX L1, CX L3 through CX L31, and CX L34 in their own right. These articles in the aggregate show that there is at least a perception of environmental concerns resulting from toxic waste and pollution as well as pulmonary problems in both Kazakhstan in general and in the city of Almaty in particular. Although the source of some of the articles is still unclear, I do not question that the Internet articles submitted by the Claimant were obtained from a valid Internet search as described in Ms. Shepard's affidavit. With that being said, Employer is quite correct that the articles lack foundation and have not been properly authenticated (even though I have no reason to question their authenticity), and some of them contain hearsay or double hearsay. Certain of the websites, such as the website maintained by the United Nations and, to a lesser extent, related links (including the United Nations Development Programme and the United Nations Environment Programme), carry an indicia of reliability, while others, such as that maintained by the so-called Greenwomen (which site apparently can no longer be accessed) are from unknown, questionably reliable sources. In particular, I find that the epidemiological study supposedly conducted by the Ministry of Health in Kazakhstan lacks probative value as there is no basis for concluding that such a study, if it actually were conducted, has been accurately reported by the Greenwomen. The report does not appear in a medical journal, peer reviewed or otherwise, but is in a paper prepared by journalists who are apparently also

environmental activists, from the tenor of the article. As no actual data from the study has been reported, it is also impossible to determine whether appropriate controls were applied or to assess the import of the findings. As Dr. Friedman has testified, the Greenwomen article is not a scientific paper, it was produced by an advocate group, and it has not been peer reviewed. (EX E2 at 34-35). Thus, CX L1 is entitled to no weight. Of the other articles, those from the United Nations website (www.un.org) and certain of its links (specifically, CX L3, CX L6, CX L10, CX L14 [excerpt from CX L6], CX L15, CX L17 [excerpts from CXL15], CX L29) warrant some consideration, although they are admittedly hearsay documents not subject to cross examination. While I continue to find that all of the articles are at least admissible to the extent that they may form a basis for Dr. Wagner's opinion, most of them have little if any probative value in their own right.

Turning to the articles annexed to Dr. Friedman's deposition, I find that they are entitled to some weight, both individually and to the extent that they have been considered by the medical experts. In this regard, both expert witnesses relied upon them without questioning their validity; at least the first of these six articles is from a peer-reviewed journal (according to Dr. Friedman's testimony); and they are all from medical or scientific journals. Although these articles have some probative value in their own right, I primarily find them of significance to the extent that they support the testimony of the expert witnesses.

Discussion

Summary of Law and Rationale from Previous Decision

In the previous decision, I summarized the pertinent law. That summary appears below:

Establishment of Compensable Injury

> According to the Act, an injury is defined as an "accidental injury or death arising out of and in the course of employment." 33 U.S.C. § 902(2). Under section 20(a), 33 U.S.C. § 920(a), it is presumed, in the absence of substantial evidence to the contrary, that a claim comes within the provisions of the Act. However, the presumption does not assist Claimant in establishing a prima facie case, which must be established before invoking the presumption. ***Devine v. Atlantic Container Lines, G.T.E.***, 23 BRBS 280 (1990). "[A] prima facie 'claim for compensation,' ... must at least allege an injury that arose in the course of employment as well as out of employment." ***U.S. Industries/Federal Sheet Metal v. Director, OWCP (Riley)***, 455 U.S. 608, 615, 14 BRBS 631, 633 (1982). As a general rule, in order to establish a prima facie case that injury arose out of employment, a claimant must establish that (1) the claimant sustained some physical harm and (2) working conditions existed, or an accident occurred, which could have caused the harm. ***See, e.g., Adams v. General Dynamics Corp.,*** 17 BRBS 258, 260 (1985). The theory of causation must be more than "mere fancy."

Stevens v. Tacoma Boatbuilding Co., 23 BRBS 191 (1990). ***See also Champion v. S & M Traylor Bros.,*** 690 F.2d 285 (D.C. Cir 1982); ***Wheatley v. Adler,*** 407 F.2d 307 (DC Cir. 1968). After the prima facie case is established, a presumption arises under section 20(a) that the employee's injury or death arose out of his or her employment.

Here, Claimant alleges that she sustained some harm (diarrhea and shortness of breath while overseas, leading to pulmonary fibrosis) and potentially causative working conditions (her exposure to environmental pollutants, as evidenced by her observation of a cloud of dust over Almaty and State Department literature concerning pollution in Kazakhstan as a whole). For the reasons set forth below, I find that Claimant's allegations go beyond "mere fancy."

There are several Defense Base Act cases that address the issue of a claimant being exposed to certain risks overseas that he or she would not have otherwise encountered. In ***O'Leary v. Brown-Pacific Maxon, Inc.,*** 340 U.S. 504, 507 (1951), when an employee drowned while attempting a rescue in a recreational area for employees in Guam, the Supreme Court found that the Act applied and stated: "All that is required is that the 'obligations or conditions' of employment create the 'zone of special danger' out of which the injury arose." However, in ***Gillespie v. G.E. Co.,*** 21 BRBS 56 (1988) ***aff'd mem.*** 873 F.2d 1433 (1989 1st Cir.), the Benefits Review Board found that, where no evidence showed that the activity causing death (asphyxiation during autoerotic activity) was related to conditions created by the overseas job (notwithstanding the administrative law judge's finding that he was engaged in recreational activity due to separation from his spouse and family), the "zone of special dangers" test was not met. Reading these cases together, it would appear that injuries resulting from Claimant's exposure to air pollution while overseas would be covered while injuries due to her smoking would not be covered, even if the illness were first manifested while she was overseas.

As Claimant has noted, the "zone of danger" test and/or the section 20(a) presumption has been applied to establish entitlement to benefits when an employee contracted diseases overseas or as a result of exposure overseas. ***See, e.g., Travelers Insurance Company v. Donovan,*** 221 F.2d 886 (D.C. Cir. 1955) (Red Cross employee with tuberculosis resulting from exposure in Kyoto, Japan).

There are also several Longshore cases that specifically address the issue of occupational disease resulting from exposure to toxic substances in the work place. Typically, such a case involves exposure to a recognized toxic substance (such as asbestos) and a disease or condition etiologically related to that substance (such as asbestosis or lung cancer). ***See, e.g., Kiev v. Bethlehem Steel Corp.,*** 16 BRBS 128 (1984) (where asbestos exposure and cancer established, presumption

found to be applicable but rebutted); ***Romeike v. Kaiser Shipyards,*** 22 BRBS 57 (1989) (pleural plaques caused by asbestos exposure found to constitute "harm"). While the instant case is somewhat unusual, in that Claimant has not identified a specific etiological agent that she claims to have caused her pulmonary fibrosis, some analogous cases have found the presumption to be applicable:[28]

1) In ***Janusziewicz v. Sun Shipbuilding & Dry Dock Co.,*** 22 BRBS 376, 1989 WL 245314 (1989), the Benefits Review Board ("Board") found that the claimant had established a prima facie case, sufficient to invoke the presumption, by showing exposure to industrial pollution at work (dust, fumes and smoke from welding, sandblasting and painting) and a respiratory impairment (chronic chest congestion and shortness of breath, reflecting an aggravation of a preexisting pulmonary condition). There was no rebuttal.

2) In ***Devine v. Atlantic Container Lines, G.I.E.,*** 23 BRBS 279, 1990 WL 284049 (1990), a prima facie case was established by a showing of exposure to certain toxic chemicals (including T-amylamine and PCB's which were leaking from broken drums in a shipyard and creosote treated telephone poles stored on the docks) and a doctor's testimony and report opining that the claimant's cancer of the distal bile duct and papilla of Vater were work related. However, the Board vacated the administrative law judge's finding that rebuttal had not been established by the opinions of two doctor finding the link to be improbable based upon their professional assessment of the current available scientific evidence.

3) In ***Sinclair v. United Food & Commercial Workers,*** 23 BRBS 148, 1989 WL 245251 (1989), the presumption was applied to the issue of causation when the claimant, a commercial artist, alleged that her exposure to chemicals in the work place (such as n-hexane in Bestine, a thinner) caused her to experience headaches, fatigue, chest and stomach pains, dizziness, and other symptoms and aggravated her preexisting psychiatric condition, making it impossible for her to work around chemicals. The Board found the presumption to apply to the psychiatric condition as well as the physical symptoms and held that the claimant did not need to prove a causal connection between the physical symptoms and aggravation of her psychiatric condition; all she needed to show was "the existence of working conditions which could conceivably cause the harm alleged." The Board also affirmed the finding that the presumption had not been rebutted.

4) In ***Stevens v. Tacoma Boatbuilding Co.,*** 23 BRBS 191, 1990 WL 284079 (1990), the Board found the presumption applicable where the claimant alleged that the decedent's exposure to paint chemicals (specifically, Tributylten

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[28] Certain of these cases applied the now defunct "true doubt" rule (***see Director, OWCP v. Greenwich Collieries***, 512 U.S. 267, 28 BRBS 43 (CRT) (1994)).

[TBT]) lowered his resistance to disease, based upon the testimony of an occupational medicine specialist, and led to a rare degenerative brain disease (Jakob-Creutzfeldt disease), which has a viral etiology, by making him more susceptible to the disease or hastening its development. The Board found that this theory went beyond "mere fancy" and that it did not need to address the rebuttal issue in view of its finding that "the administrative law judge correctly weighed the evidence of record, properly resolved the close question of causation in favor of claimant, and, thus, properly determined that claimant is entitled to compensation benefits under the Act due to decedent's death."

5) In ***Peterson v. Columbia Marine Lines,*** 21 BRBS 299, 1988 WL 232763 (1988), the Board agreed with the administrative law judge that the presumption was applicable when a claimant developed harm (chest pain) and alleged hypersensitivity caused by exposure to a variety of chemicals (including DD soil fumigant, vapom, telon 2, telon 7, fertilizers, and gasoline) over a period of years, supported by an allergist's opinion, even though the doctor could not identify the specific chemicals which produced his hypersensitivity. The Board went on to affirm the administrative law judge's finding that, although the presumption had been rebutted, the claimant had established that his disability was work related based upon all the evidence.

6) In ***Stevens v. Todd Pacific Shipyards,*** 14 BRBS 626 (1982), in a split decision, the Board found the presumption to be applicable to a grinder/burner who was exposed to high concentrations of various industrial dust particles and later developed industrial bronchitis (which resolved) and sarcoidosis, even though the sarcoidosis was deemed to be of unknown etiology. The majority found that the employer's proof, which consisted of expert medical opinion that the cause was unknown and proof that the disease occurs naturally, fell short of establishing rebuttal.

See also Champion v. S & M Traylor Bros., 690 F.2d 285, 15 BRBS 33 (D.C. Cir. 1982) (emotional trauma and aggravation of preexisting asthma by exposure to dust and fumes from construction in a subway tunnel); ***Woodside v. Bethlehem Steel Corp.,*** 14 BRBS 601 (1982) (decedent, a painting specialist, had chronic obstructive pulmonary disease which may have hastened his death and had been exposed to various substances at work which could have caused it). The preponderance of cases clearly suggest that the presumption is broad and may be invoked notwithstanding uncertainty as to the exact hazardous substance involved when a claimant is exposed to more than one toxic substance. They also show that, where a disease has a viral etiology, exposure to a toxic agent which facilitates infection with the virus is sufficient to invoke the presumption.

On the other hand, there are cases which suggest a more stringent burden for claimant to establish occupational exposures which could have caused the alleged harm. In ***Wendler v. American National Red Cross,*** BRB No. 93-0423 (May 29, 1996) (unpublished),[29] the Board affirmed the administrative law judge's finding of no prima facie case when the claimant alleged chemical hypersensitivity due to Agent Orange exposure but failed to show proof of agent orange exposure or any objective physical symptomatology showing herbicide exposure when she was employed in Korea for the Red Cross. Of note is the fact that the claimant was able to show that there was Agent Orange in Korea at the time she was employed there but her testimony that she was present at the location in Korea where the Agent Orange was applied was found to be not credible. Similarly, in ***Lacy v. Four Corners Pipe Line,*** 17 BRBS 139, 1985 WL 5352 (1985), where the claimant developed hepatitis allegedly caused by toxic chemical exposure (which made her susceptible to the hepatitis virus), the Board remanded for a determination whether claimant had met her burden of establishing exposure to potentially toxic chemicals during the latent period for the disease. The significance of that decision is the requirement that the claimant not only establish exposure to a potentially causative toxic agent but also show that such exposure was within the recognized latent period for the disease. Also, in ***Blue v. CR Industries,*** 1989-LHC-2564 (May 1, 1992), the administrative law judge found there was no acceptable medical evidence to support a finding of a relationship between fatty tissue buildup and bulk phosphate fertilizers, despite claimant's allegation of exposure to various chemicals and compounds.

In applying these principles, I found that the Claimant had established a basis for the invocation of the section 20(a) presumption, because she proved "that she sustained some harm (pulmonary fibrosis first manifested as shortness of breath) and potentially causative working conditions (exposure to air pollution in Almaty, Kazakhstan)." This finding was based upon Claimant's credible testimony that she was exposed to a "cloud of dust" by her assignment in Almaty combined with "the testimony of her treating physician, Dr. Wagner, who believed that the causative agent for her pulmonary fibrosis dated to the time of her employment in Almaty, based upon her complaints of shortness of breath there, the period of time that elapsed before she complained of acute symptomatology, and the degree of active inflammation present in Claimant's lungs at the time of diagnosis" and his opinion "that there was a causal relationship between the Claimant's exposure to air pollutants in Almaty, Kazakhstan and her later development of pulmonary fibrosis."

I also discussed the law relating to rebuttal of the section 20(a) presumption:

Rebuttal of Presumption

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[29] This decision is available through the Benefits Review Board link on the Office of Administrative Law Judges website (www.oalj.dol.gov).

Once this presumption is invoked, the burden shifts to the employer to rebut the presumption with substantial countervailing evidence which establishes that the claimant's employment did not cause, contribute to, or aggravate his or her condition. ***James v. Pate Stevedoring Co.***, 22 BRBS 271 (1989); ***Peterson v. General Dynamics Corp.***, 25 BRBS 71 (1991). "Substantial evidence" means evidence that reasonable minds might accept as adequate to support a conclusion. ***E & L Transport Co. v. N.L.R.B.***, 85 F.3d 1258 (7th Cir. 1996). Employer must produce facts, not speculation, to overcome the presumption of compensability. Reliance on mere hypothetical probabilities in rejecting a claim is contrary to the presumption created by section 20(a). ***See Smith v. Sealand Terminal***, 14 BRBS 844 (1982). Rather, the presumption must be rebutted with specific and comprehensive medical evidence proving the absence of, or severing, the connection between the harm and employment. ***Hampton v. Bethlehem Steel Corp.***, 24 BRBS 141, 144 (1990). When aggravation of or contribution to a pre-existing condition is alleged, the presumption still applies, and in order to rebut it, Employer must establish that Claimant's condition was not caused or aggravated by his employment. ***Rajotte v. Genera Dynamics Corp.***, 18 BRBS 85 (1986). In ***Stevens v. Todd Pacific Shipyards, supra***, the Board indicated that an employer could establish rebuttal by evidence "negating any potential relationship between sarcoidosis and claimant's industrial exposure," even if it could not prove an alternate cause. Once the employer has carried its burden of producing substantial evidence sufficient to justify a finding that a claimant's injuries were due to a cause unrelated to the workplace, the presumption is rebutted and is dropped from the case. ***American Grain Trimmers, Inc. v. OWCP,*** 181 F.3d 810 (7th Cir. 1999) ***cert. denied,*** 528 U.S. 1187 (2000).

The Benefits Review Board discussed the issue of what constitutes adequate rebuttal in ***O'Kelley v. Dept. of the Army/NAF***, 34 BRBS 39 (2000), a case arising in the Eleventh Circuit, and found that substantial evidence was enough, even though the Board stated that the Eleventh Circuit "espoused a 'ruling out' standard when addressing the issue of rebuttal of the Section 20(a) presumption" (citing ***Brown v. Jacksonville Shipyards, Inc.,*** 893 F.2d 294, 23 BRBS 22 (CRT) (11th Cir. 1990)). ***O'Kelley*** involved a golf course worker exposed to herbicides, fungicides and insecticides, who claimed aggravation of a neurological disorder. The employer's expert opined within a reasonable degree of medical certainty that the claimant's condition was neither caused by nor contributed to by his exposure to chemicals while working for the employer, but on cross examination he conceded that it was "possible" that the claimant's condition was work-related and he offered no opinion as to the cause for the worsening of claimant's condition. The Board found that the administrative law judge erred in finding this rebuttal to be inadequate. In so finding, the Board observed that "absolute certainty" is a difficult concept in the medical profession, and as the expert's reports and testimony "unequivocally express[ed] his opinion,

> rendered within a reasonable degree of medical certainty, that [the] claimant's condition [was] not work related," the employer had produced "evidence sufficient to sever the causal relationship between [the] claimant's employment and his harm." ***Compare Jones v. Aluminum Company of America,*** 2001 WL 467885 (BRB April 9, 2001) (testimony indicating that cancer was caused by a combination of two risk factors, with cigarette smoking as greatest risk factor with asbestos exposure as lessor risk factor, insufficient to establish rebuttal of asbestos as cause.)

Applying these principles, I found that the Employer had established rebuttal based upon the deposition testimony and report of its expert, Dr. Friedman, who "opined that Claimant's exposure to industrial pollutants did not cause her pulmonary fibrosis, because exposure to toxic agents has been shown to be a risk factor for idiopathic pulmonary fibrosis in a work setting, but not based upon a casual street or home exposure," indicated that Claimant "would have had a history of industrial bronchitis while in Almaty if her exposure to pollutants were the causative agent," "suggested that the onset would have been more gradual," and "concluded that he had ruled out Claimant's environmental exposure as a cause of her pulmonary fibrosis." I therefore found that the Employer had met its burden of production on the rebuttal issue.

As I also noted, establishment of rebuttal brought the presumption out of the case and required that the causation issue must be addressed on the merits:

> **Merits of the Causation Issue**
>
> As noted above, once the employer has carried its burden of producing substantial evidence rebutting the nexus between a claimant's injuries and the workplace, the presumption is rebutted and is dropped from the case. ***American Grain Trimmers, Inc. v. OWCP, supra.*** It then becomes the burden of a claimant to establish causation by a preponderance of the evidence, as the claimant would be required to do for any of the necessary elements of the claim. ***See Director, OWCP v. Greenwich Collieries***, 512 U.S. 267, 28 BRBS 43 (CRT) (1994) (invalidating the "true doubt" rule, which gave the benefit of the doubt to claimants).

In my original decision, I found that the Claimant had failed to sustain this burden. I concluded that, at bottom, Claimant's claim relied upon speculation and she had not established causation by a preponderance of the evidence.

Basis for Modification

The case before me involves a petition for modification under the Act. In this regard, section 22 of the Act provides, in relevant part:

922. Modification of awards

> Upon his own initiative, or upon the application of any party in interest (including an employer or carrier which has been granted relief under section 8(f) [33 USC §§ 908(f)]), **on the ground of a change in conditions or because of a mistake in a determination of fact** by the deputy commissioner [district director], the deputy commissioner may, at any time prior to one year after the date of the last payment of compensation, whether or not a compensation order has been issued, or at any time prior to one year after the rejection of a claim, review a compensation case (including a case under which payments are made pursuant to section 44(i) [33 USC §§ 944(i)]) in accordance with the procedure prescribed in respect of claims in section 19 [33 USC §§ 919], and in accordance with such section issue a new compensation order which may terminate, continue, reinstate, increase, or decrease such compensation, or award compensation. . . . [Emphasis added.]

33 U.S.C. §922.[30] ***See also*** 20 C.F.R. § 702.373. Review of a compensation case for the purpose of modification is available only upon the grounds of a change in conditions or a mistake in determination of fact. 20 C.F.R. § 702.373(c). Modification of a final decision may be based on a mistake of fact in the initial decision or a change in claimant's economic or physical condition at any time prior to one year after the last payment of compensation or the rejection of the claim. ***See Metropolitan Stevedore Co. v. Rambo***, 515 U.S. 291, 30 BRBS 1 (CRT) (1995); ***Fleetwood v. Newport News Shipbuilding & Dry Dock Co.***, 776 F.2d 1225, 18 BRBS 12 (CRT) (4th Cir. 1985); ***Finch v. Newport News Shipbuilding & Dry Dock Co.***, 22 BRBS 196 (1989). While modification may not be used to correct errors of law, it is applicable to mixed questions of fact and law, which are treated as mistakes of fact. ***McDougall v. E.P. Paup Co.***, 21 BRBS 204 (BRB 1988). ***See also Presley v. Tinsley Maintenance Service***, 529 F.2d. 433 (5th Cir. 1976).

An administrative law judge has wide discretion to modify a compensation order. ***O'Keeffe v. Aerojet-General Shipyards, Inc.***, 404 U.S. 254, 256 (1971) (***per curiam***); ***Bath Iron Works v. Director, OWCP [Hutchins]***, 244 F.3d 22 (1st Cir. 2001) (both decided under Longshore and Harbor Workers' Compensation Act)). However, it is also true that the modification procedures may not be used as a "back-door route to retry" the previous case. ***Kinlaw v. Stevens Shipping and Terminal Co.,*** 33 BRBS 68 (BRB 1999) (Longshore case). Modification on the grounds of a mistake in determination of fact may be based upon an allegation that the ultimate fact was mistakenly decided; "[t]here is no need for a smoking-gun factual error, changed conditions, or startling new evidence." ***Jessee v. Director, OWCP***, 5 F.3d 723, 725 (4th Cir. 1993) (Black Lung decision).

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[30] The term "deputy commissioner" as used in the Act has been replaced by the term "district director." 20 C.F.R. § 702.105.

In ***O'Keeffe***, which involved reopening of a Longshore case based upon new testimony by the claimant's treating physician and another doctor, the Court of Appeals had held that in the absence of changed conditions or new evidence clearly revealing a mistake, the deputy commissioner lacked the authority to change his mind based upon additional evidence that was cumulative in nature. Noting that neither the wording of the statute nor its legislative history supported this "narrowly technical and impractical construction," the Supreme Court stated in ***O'Keeffe***:

> Thus, on its face, the section permits a reopening within one year 'because of a mistake in a determination of fact.' There is no limitation to particular factual errors, or to cases involving new evidence or changed circumstances. The Act at one time did authorize reopening only on the 'ground of a change in conditions,' 44 Stat. 1437, but was amended in 1934 expressly to 'broaden the grounds on which a deputy commissioner can modify an award * * * when changed conditions or a mistake in a determination of fact makes such modification desirable in order to render justice under the act.' S.Rep.No.588, 73d Cong., 2d Sess., 3--4 (1934); H.R.Rep.No.1244, 73d Cong., 2d Sess., 4 (1934). **The plain import of this amendment was to vest a deputy commissioner with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted.** [Emphasis added].

404 U.S. at 255-56.

Here, although new evidence has been submitted, the gravamen of Claimant's petition is that consideration of the newly submitted evidence warrants a finding of mistake in determination of fact. Specifically, in the Motion for Modification of Decision and Order Denying Benefits filed with the Benefits Review Board on September 19, 2001, Claimant asserts the following:

> . . . Claimant's motion is supported by newly discovered evidence which describes in detail the toxic environmental conditions in Almaty, Kazakhstan where she lived and worked as part of her overseas employment and more significantly identifies a specific pollutant within Almaty, i.e., Cadmium, that is known to trigger pulmonary fibrosis. Based on this new evidence, claimant submits that she has overcome the ALJ's concern that she has failed to present specific evidence of the environment in Almaty and more specifically the presence of a specific toxic agent that may have caused the development of her work-related illness.

Id. at 1.[31] Claimant further asserts that the new evidence (which includes the articles and affidavits discussed above) is "necessary to the fair adjudication of her claim." ***Id.*** at 2.

As noted above, Claimant's original claim for benefits under the Longshore and Harbor Workers' Compensation Act was denied by the undersigned's Decision and Order issued May 24, 2001. In particular, that decision found that while the Claimant had established a ***prima facie*** case, the Employer had produced substantial evidence which rebutted the presumption, warranting consideration of the claim on the merits. Ultimately, the claim was denied because of Claimant's failure to prove by a preponderance of the evidence that her pulmonary condition arose from her employment in Almaty, Kazakhstan given the inability of Claimant to identify a specific etiological agent responsible for her condition, coupled with the testimony of Employer's expert witness as to his opinion that the Claimant's condition was caused by a viral agent rather than a toxic exposure. Thus, the thrust of the new evidence submitted by Claimant focuses on the identification of an etiological agent responsible for Claimant's condition, namely, Cadmium. However, the new evidence also has a bearing upon the original theory espoused by Claimant, that her exposure to environmental pollutants in Almaty gave rise to her pulmonary fibrosis.

In reviewing the newly submitted evidence, I find, as I did before, that she has made a ***prima facie*** case that her pulmonary fibrosis was caused by her exposure to pollutants in Almaty. After having reviewed Dr. Wagner's explanation of what the term "usual interstitial pneumonitis" means, I no longer place any significance upon whether Claimant now satisfies the definitional criteria for the condition. On the issue of whether cadmium was the culprit, I find that the evidence submitted by Claimant falls short of establishing that she was exposed to potentially harmful levels of cadmium, although she has raised the possibility.

Turning to the issue of rebuttal, I note that the rebuttal presented in the instant case is somewhat weaker than that originally submitted. Specifically, Employer's expert witness, Dr. Friedman, has backed off somewhat from the position that the Claimant would have manifested symptoms of bronchitis (such as a cough) if her pulmonary fibrosis were due to environmental pollutants, and he also did not place as much reliance upon the relatively short period of time that it took for symptoms to develop. (EX E2 at 76 to 77).[32] Although noting that bronchitis could

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[31] Although it is only on modification that the Claimant has focussed upon cadmium as an etiological factor, at least one of the articles submitted at the previous hearing also mentioned cadmium. That article reported cadmium poisoning among children in a town on the Russian border to Kazakhstan, allegedly caused by a copper smelting plant that was no longer in operation. (***See, e.g.,*** CX D at 34.) When asked about that article at his previous deposition, Dr. Friedman testified that "Cadmium, per se, can cause pulmonary fibrosis." (CX K p. 45).

[32] The following is from Dr. Friedman's April 15, 2002 deposition:

> Q. Doctor, in your earlier testimony you indicated and the court recognized it, she stated, "Furthermore, Dr. Friedman indicated that Ms. Cohen would have had a history of industrial bronchitis while in Almaty if her exposure to pollutants were the causative agent and he suggested the onset would have been more gradual."

be associated with either pollution or smoking, he did not seem to place much significance upon it as a symptom at his most recent deposition. ***Id.*** at 75 to 76. He also backed off on the statement that the onset would have been more gradual in the event that the lung biopsy showed mature scarring (which, in fact, it did), as such scarring (as contrasted with immature scarring) would not be expected to occur within a six-day period. ***Id.*** at 66-67. Although Dr. Friedman continued to maintain that it was unlikely that environmental pollutants were a factor, based upon assumptions he made as to the likely amount of Claimant's exposure, he did not rule them out as a cause. On the issue of cadmium, Dr. Friedman opined that short term, acute cadmium poisoning was unlikely to have been the cause of Claimant's pulmonary fibrosis, in view of the absence of the distinctive symptoms of acute cadmium toxicity. With respect to chronic cadmium exposure, he felt that the exposure levels in an outside setting (as opposed an indoor work place) would be too low and the time period of exposure (seven months) would be too short. He also noted that mature fibrosis was not reversible and would not respond to steroids. However, he did not so much rule out cadmium as believe it to be an unlikely cause, in view of the atypical circumstances, and he conceded that "[e]verybody reacts differently." (EX E2 at 76). Although the alternate cause suggested by Dr. Friedman – a viral etiology – was ruled out by Claimant's treating physician based upon the bronchial lavage, it is not necessary for the rebuttal to show another etiologic agent for it to constitute substantial evidence. ***See Stevens, supra.*** Based upon consideration of the rebuttal evidence, I find that Dr. Friedman's deposition testimony and report, if credited, would support a finding that it is unlikely that the Claimant's exposure to environmental pollutants in general, and cadmium in particular, caused, contributed to, or aggravated her condition. However, Dr. Friedman's testimony does not rule out either environmental pollutants or chronic cadmium exposure as etiological factors, and he has readily admitted his lack of information as to Claimant's actual exposure and her specific clinical findings.[33] Moreover, his recent equivocation, coupled with his lack of knowledge as to Claimant's medical records and the specific facts surrounding her exposure to pollutants in Almaty, makes it questionable that his opinion constitutes "substantial countervailing evidence" so

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> A. I don't know what that means. I'm just saying that exposure to general pollution can cause acute bronchitis. Acute bronchitis can lead to chronic bronchitis. But that doesn't allow us to diagnose interstitial fibrosis which in an industrial setting, acute cadmium toxicity would be associated with fibrosis. If that has to be a modification, then it is. That's my opinion.

(EX E2 at 76-77.)

[33] When asked whether it would be unusual for there to be an industrial level of cadmium in an outdoor environmental setting, Dr. Friedman stated:

> A. Nor under any ordinary circumstances. It's usually an indoor setting where there is poor ventilation and airflow and air exchange, yet, but an outdoor setting, that would rarely happen. Maybe in a severe inversion where there is a tremendous amount of pollution and it stays steady and unchanged. It's a possibility but not necessarily so.

EX E2 at 80-81. As discussed above, Claimant alleges that there was an inversion in Almaty and she claims that the surrounding mountains trapped in the air, causing the city to appear as a "cloud of dust" and resulting in black ice which remained frozen during the winter.

as to constitute rebuttal. ***See American Grain Trimmers, Inc. v. OWCP,*** 181 F.3d 810 (7th Cir. 1999) ***cert. denied,*** 528 U.S. 1187 (2000); ***Piceynski v. Dyncorp.***, BRB No. 97-1451 (BRB 1998)..

There is, however, another problem with the rebuttal evidence that I did not address in my initial decision, as it has only become clear in light of the evidence now before me. Specifically, at his ***de bene esse*** deposition in the instant case, when Dr. Friedman opined that a virus was most likely the cause of Claimant's pulmonary fibrosis, he went on to explain that such a virus would have led to acute lung injury within a matter of days from her contraction of the virus, and he was aware of a case involving a ten-day period between exposure and onset of symptomatology.[34] (EX E2 at 23-24). When told that the biopsy revealed the presence of mature fibrosis, immature fibrosis, and active inflammation six days after Claimant returned to the United States, he indicated that she could have contracted the virus when she was in Almaty, and the virus could have been responsible for the diarrhea that she complained of when she was there. (EX E2 at 71 to 72). In my initial decision, I found Dr. Friedman's suggestion that a virus was responsible to be more plausible based upon his discussion of the evidence. At that time, there was no evidence showing whether the latent period would place Claimant's contraction of the virus at the time that she was in Almaty. However, if Dr. Friedman is correct that a virus is responsible, his recent ***de bene esse*** deposition establishes that most likely she contracted the virus when she was in Almaty. The rebuttal does not, therefore, constitute "evidence sufficient to sever the causal relationship between [the] claimant's employment and [her] harm." ***See O'Kelley v. Dept. of the Army/NAF***, 34 BRBS 39 (2000). The inadequacy of the rebuttal is particularly clear in the instant case, in view of the applicability of the "zone of danger" test, which gives rise to liability based upon a claimant's activities outside of work in a foreign country. ***See O'Leary v. Brown-Pacific Maxon, Inc.,*** 340 U.S. 504, 507 (1951). Quite simply, the testimony of the expert witnesses on both sides tends to support a finding that the Claimant's pulmonary condition arose out of her sojourn in Almaty.

It is also worth noting that both physicians expressing opinions in this case have opined that whatever the cause of Claimant's pulmonary fibrosis, it arose when she was in Kazakhstan and she is no longer exposed to the causative agent.[35] As Dr. Wagner pointed out, the biopsies revealed an ongoing process that had continued for a period of months and was still continuing at the time of Claimant's hospitalization, and Dr. Friedman essentially agreed when the biopsy results were given to him. Dr. Wagner also noted that when Claimant was removed from the hazardous environment of Kazakhstan and placed on steroids, the active portion of her condition resolved, although the residual fibrosis will never go away. Even though Claimant resumed

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[34] I n contrast, Dr. Wagner indicated that the latent period for a virus could extend from a period of days to a period of years or even decades, depending upon the virus. (TR2 at 218 to 219.)

[35] Parasites, antibiotic treatment, and drug therapy were other suggested etiologies for the disease that could have arisen in Almaty, but neither physician found them to be causative agents in the instant case. (***See, e.g.,*** CX J pp.92 to 95, 103 to 106; CX K pp. 7 to 11, 17 to 20, 76, 79 to 80).

smoking and stayed on the same medications, the active process of the disease did not resume. Thus, the evidence shows that factors in the "zone of danger" gave rise to her disease, regardless of whether it was an environmental exposure, a virus, or some other factor.

Accordingly, I find that there is a basis for modification based upon a mistake in determination of fact on the rebuttal issue. Specifically, I now find that the rebuttal was inadequate to sever the causal connection between Claimant's employment in Almaty and her pulmonary fibrosis. It is therefore necessary for me to reopen the case and consider it on the merits.

In reaching this conclusion, I note that even if the rebuttal were deemed to be adequate, there would be a basis for modification based upon my finding that a preponderance of the evidence now supports the claim that environmental pollutants were the cause of Claimant's pulmonary condition. This matter will be discussed ***infra.***

Merits of the Case

Causation

On consideration of the merits, I find, for the reasons set forth above, that the Claimant has established that her pulmonary condition presumptively arose out of her employment in Almaty, and I further find that the rebuttal adduced by the Employer is insufficient to sever the causal relationship. I make this finding based upon her exposure to environmental pollutants in Almaty, which has been established. While it is certainly possible that cadmium was responsible, there is not enough good quality evidence of her exposure to cadmium for Claimant to invoke the section 20(a) presumption. As Claimant has presumptively established that her pulmonary condition was caused by her exposure to environmental pollutants in Almaty, and causation has not been rebutted, she has established her entitlement to benefits.

Assuming, ***arguendo***, that the rebuttal were deemed to be adequate, it would be necessary for the evidence as a whole to be considered on the issue of causation. I will now do so in the interest of completeness. Thus, to the extent that the rebuttal may be deemed to be adequate, I find that there was a mistake in determination of fact on the causation issue overall, so as to give rise to consideration of the claim on the merits, and a grant of benefits, for the reasons set forth below.

In my original decision, I found that the Claimant had failed to sustain her burden of proof based upon her failure to identify one or more causative agents which were the likely cause of her pulmonary fibrosis, the uncertainty of the diagnosis (which would not satisfy the current criteria for UIP), the equivocation used by Claimant's treating physician (Dr. Wagner) in stating his opinion, and the deposition testimony by Dr. Friedman which called into question Dr. Wagner's analysis. However, based upon the newly submitted evidence, taken into consideration along with the evidence previously of record, I find that Claimant has satisfied her burden of establishing that

her pulmonary condition arose from her employment in Almaty. Specifically, Dr. Wagner has now identified a potential causative agent (cadmium) and, while Claimant's exposure to that agent is unclear, its identification provides further support to his opinion, and his discussion of its possible effect was enlightening on the issue of toxic exposures in general. Dr. Wagner's testimony has explained that "UIP" was a description of findings as opposed to a diagnosis and he has now stated his opinion without equivocation, to a reasonable degree of medical certainty (***i.e.***, in terms of probability). In contrast, Dr. Friedman's testimony is now stated with some uncertainty, and the other possible etiology he has suggested is one that would also result in an award of benefits.

Upon consideration of all of the evidence of record, I find that Dr. Wagner's testimony is the most persuasive and, while the evidence upon which he relies is not of sufficient probative value to establish Claimant's exposure to cadmium, his opinion establishes that exposure to environmental pollutants in Almaty was most likely the cause of Claimant's problems.[36] In this regard, Dr. Wagner has ruled out the other identified etiological factors. Dr. Wagner indicated, contrary to Dr. Friedman, that he could rule out both viruses and bacteria as causative agents based upon pathological evidence from the bronchial wash, as later confirmed by biopsy.[37] Both he and Dr. Friedman indicated that smoking was a risk factor that increased Claimant's susceptibility to pulmonary fibrosis and not a causative agent, and Dr. Wagner pointed out that her condition did not worsen when she resumed smoking. Dr. Wagner explained that none of the antibiotics, antidepressants, or other medicines that Claimant was taking have been associated with pulmonary fibrosis, and she was worked up for collagen vascular disease (such as systemic lupus erythematosus) without positive findings.

Dr. Wagner also pointed to a number of factors that placed the cause of Claimant's problems as her employment in Almaty. Dr. Wagner found it to be significant that her condition resolved, in contrast to most cases of IPF, which lead to death, a distinction that he attributed to the fact that Claimant was removed from the causative agent. In view of the absence of honeycombing of the lungs on a high resolution CT scan and the mature scarring, which reflected an insult that had occurred within the past three or four months, Dr. Wagner placed the onset of Claimant's pulmonary condition as February or March, 1998, consistent with Claimant's symptoms of shortness of breath and exercise intolerance. The presence of active disease at the time of her admission indicated that the process was continuing. Although disagreeing as to the cause, Dr. Friedman essentially agreed as to the time of the insult. Exposure to toxic substances was the only factor that had not been ruled out, and based upon the Claimant's clinical data, the epidemiologic studies cited by Dr. Friedman, and the other articles obtained by Claimant, Dr.

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[36] Dr. Wagner focussed on cadmium rather than other possible etiologic agents because the pattern fit as compared with other possible pollutants, such as leaded gasoline. (TR2 at 198 to 199).

[37] The results of the bronchial wash appear at CXG2, pp. 855-56. Reports of the bronchial lung biopsy and wedge (open lung) biopsy appear, ***inter alia***, at CXG2 pp. 848-50.

Wagner found that her exposure to pollution in Almaty was the most likely cause of her pulmonary condition, and that cadmium was the most likely toxic substance.

In weighing the probative value of the opinions, I have taken into consideration the fact that some of the articles relied upon by Dr. Wagner are of questionable probative value. Nevertheless, his opinion is still more persuasive than that of Dr. Friedman, even if the supporting documentation is discounted, and I found him to be a very credible witness. It was also invaluable that I had an opportunity to question him to obtain a better understanding of his opinion. Although Dr. Friedman also appeared to be credible, his opinion is undermined by his lack of knowledge as to Claimant's clinical data, such as the specific biopsy findings. In contrast, as Claimant's treating physician during the critical early stages of her disease, Dr. Wagner is intimately aware of the particulars of her condition. Dr. Wagner's opinion is both well reasoned and documented. In addition, Dr. Wagner has superior credentials to those of Dr. Friedman for the purpose of stating an opinion as to the etiology of Claimant's pulmonary disease, as he possesses the additional credentials of board certification in pulmonary diseases and critical care medicine, both of which disciplines impact the instant case. Accordingly, I find that Dr. Wagner's opinion outweighs that of Dr. Friedman, and I further find that Claimant has established that her pulmonary condition arose out of her exposure to environmental pollutants during the course of her employment in Almaty.

Compensable Injuries

According to the Act, an injury is defined as an "accidental injury or death arising out of and in the course of employment." 33 U.S.C. § 902(2). Aggravation of a preexisting injury may be compensable by virtue of the section 20(a) presumption. ***See Port of Portland v. Director,*** 932 F.2d 836 (9th Cir. 1991) (defining the aggravation rule as "a doctrine of general workers' compensation law which provides that, where an employment injury aggravates, accelerates, or combines with a preexisting impairment to produce a disability greater than that which would have resulted from the employment injury alone, the entire resulting disability is compensable.") ***See also Bass v. Broadway Maintenance***, 28 BRBS 11 (Feb. 25, 1994) [available at BRB website under 89-1360a]; ***Frye v. Potomac Electric Power Co.,*** 21 BRBS 194, 1988 WL 232738 (July 29, 1988).

As noted above, the Claimant is claiming entitlement to temporary total disability benefits based upon her incapacity due to various ailments that she claims resulted from her pulmonary fibrosis or its treatment. Temporary total disability benefits are payable in the amount of 66 2/3 per centum of a claimant's average weekly wage during the continuance of the disability. 33 U.S.C. §§ 908.

Claimant also claims medical benefits, and specifically the reimbursement for her out of pocket medical expenses and related expenses, necessitated by the treatment of the various ailments. In this regard, Section 7(a) of the Act provides that:

> The employer shall furnish such medical, surgical, and other attendance or treatment, nurse and hospital service, medicine, crutches, and apparatus, for such period as the nature of the injury or the process of recovery may require.

33 U.S.C. § 907(a). ***See also*** 20 C.F.R. Sec. 702.413. When an employer has declined to furnish medical or other treatment or services, an employee may be reimbursed for the costs of medical treatment. 33 U.S.C. § 907(d)(1).

To obtain reimbursement, a claimant must establish that the medical expenses are related to the compensable injury. ***Pardee v. Army & Air Force Exch. Serv.***, 13 BRBS 1130 (1981); ***Suppa v. Lehigh Valley R.R. Co.***, 13 BRBS 374 (1981). The employer is liable for medical services for all legitimate consequences of the compensable injury, including medical malpractice in the treatment of such injury. ***Lindsay v . George Washington University***, 279 F.2d 819, 820 (D.C. Cir. 1960); ***Austin v. Johns-Manville Sales Corp.***, 508 F. Supp. 313, 318 (D. Me. 1981). Section 7 does not require than an injury be economically disabling in order for a claimant to be entitled to medical expenses, but only that the injury be work-related. ***Frye v. Potomac Elec. Power Co.***, 21 BRBS 194 (1988). Treatment is compensable even though the injury is due only partly to a work-related condition. ***Turner v. Chesapeake & Potomac Tel. Co.***, 16 BRBS 255, 258 (1984). A claimant has established a ***prima facie*** case for compensable medical treatment where a qualified physician indicates treatment was necessary for a work-related condition. ***Id.***

Keeping the above principles in mind, I will address each of the conditions claimed by the Claimant to be related to her work-related disability or its treatment. Most of these complaints relate to Claimant's treatment with Prednisone. Dr. Wagner has described Prednisone as "not a happy drug" and he has opined that Claimant has had just about every complication attributable to Prednisone, except for cataracts. (CX J at 43).

Right and Left Hip Conditions. First, Claimant asserts that both her right and leg hips were damaged by her Prednisone treatment. Although she had planned to have a right hip replacement prior to her assignment in Almaty, she claims that it was no longer an elective procedure due to her Prednisone treatment. She had not had problems with her left hip prior to her Almaty assignment.

In the deposition that he gave in connection with the prior hearing, Dr. Wagner explained why he considered the right hip condition to have been related to or aggravated by the Prednisone treatment:

> A. Well, Prednisone can do a lot of things, but the one thing that it, one thing that it universally does in terms of bony metabolism is causes a tremendous loss of calcium. And the loss of calcium has sort of an interesting mechanism, and that is that circulating calcium level, circulating calcium is filtered by the kidney and peed out, and, as circulating calcium levels decline, the parathormone,

> parathormone levels go up, and calcium is then released from the bone, but because there's Prednisone around the released calcium is then peed out. And there is really a dramatic decrease in bone density. . . .

(CX J at 24 to 25).[38] Dr. Wagner went on to explain that the majority of bone density loss occurred in the first three months of treatment, but that the bone loss was irreparable. (***Id.*** at 25.) He concluded that "the best objective data" for his opinion that this bone loss had occurred in Claimant's case was "a radiographic report from Dr. Graeter that shows that she has actually lost about a quarter inch of her femoral height over the course of about the first two or two-and-a-half months of therapy" (***Id.***)[39] He further concluded that "her right hip is now not really an elective procedure anymore" and noted that it would have to be done on a more urgent basis (rather than next year or whenever she got around to it). (***Id.*** at 25-26.) Dr. Wagner admitted that the Claimant had preexisting arthritis and did not have avascular necrosis (as he had originally thought), but he continued to maintain that the shortening to the femoral head was attributable to Prednisone. (***Id.*** at 120 to 126.) He reached that conclusion based upon her acceleration of symptoms as well as her loss of height. (***Id.*** at 128 to 130, 135 to 137.) Dr. Wagner discussed the Claimant's right hip condition with Dr. Graeter at the time and reduced the Prednisone at his behest, so that the surgery could be performed. (***Id.*** at 130 to 132.)

When asked about the relationship between Claimant's left hip condition and replacement and her Prednisone treatment, Dr. Wagner testified:

> Q. The timing of that hip procedure has been accelerated, if she needed to have it at all. It's clear she was going to need the – well, we probably have to have her right hip done sooner or later. I think that's true. I'm not so certain she would have had to have her left hip done at all had we not whacked her with Prednisone.

(***Id.*** at 42). Dr. Wagner associated Claimant's period of temporary total disability beginning on September 9, 2000 with Claimant's Prednisone therapy, as it related to the need for her left hip repair. (***Id.***) He later explained that this opinion was based upon the "extraordinary bone loss which is associated with these doses of Prednisone." (***Id.*** at 137; ***see also*** 133 to 135).[40]

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[38] The mechanisms by which steroids can lead to osteoporosis are discussed in the article "Coping with Prednisone" (by E. Zuckerman and J. Ingelfinger) at pages 71 and 72 (CX E at 103, 105).

[39] Dr. Graeter noted in an August 19, 1998 letter that an examination of Claimant's right hip revealed "1/4 inch of shortening and range of motion limited by pain." (CX G2 at 805).

[40] In a letter of September 26, 2000, Dr. Graeter noted that an examination had shown that Claimant's left leg was "about 1 cm. shorter than the right, range of motion is painful in the groin and x-rays [s]how progression of arthritis." (CX G2 at 785A.)

Dr. Friedman disagreed with Dr. Wagner with respect to both hips. At the deposition he gave with respect to the previous case, Dr. Friedman noted that the Claimant's right hip replacement was scheduled before she went to Kazakhstan, before she was on steroids, and he attributed it to osteoarthritis in the hip. (CX K at 22, 80-81). He opined, contrary to Dr. Wagner:

> . . . . The reason for the right hip replacement was osteoarthritis in the hip. The biopsy of the hip – they took out the whole ball joint of the hip, and there was erburination and pitting. There was no evidence of avascular necrosis of the femoral head. So the steroid therapy did not affect that.
>
> The hip was something that had previously existed, and had to be replaced, and it was subsequently replaced.

(***Id.*** at 22 to 23.) Dr. Friedman opined that if the right hip condition were associated with Prednisone, there would be a vascular pattern that would be seen histologically and radiologically, which was not present in this case. (***Id.*** at 23 to 24.) He concluded that Prednisone did not augment or initiate osteoarthritis of the hip and was not part of the pathology of the osteoarthritic changes. (***Id.*** at 24 to 25.) He conceded that osteoporosis was a known risk of Prednisone, but he distinguished it from arthritis, which he explained was a joint problem, which could cause a collapse of the femoral head if it were weakened, but which does not cause pitting and erburination of the femoral head. (***Id.*** at 26.)

Upon further questioning, Dr. Friedman conceded that he had been assuming a lower dose of Prednisone, but he continued to maintain that Claimant's disease process was osteoarthritis, not avascular necrosis. (***Id.*** at 55.) When asked about the orthopedist Dr. Graeter's comment that there was flattening of the femoral head, he conceded that Dr. Graeter was concerned that Prednisone therapy could have caused it, but he continued to maintain that it was not attributable to Prednisone, as only osteoarthritis was present after the surgery. (***Id.*** at 56 to 59.) Dr. Friedman indicated that he was not an orthopedic specialist, however, and he could not comment upon the significance of the size measured of the femoral head. (***Id.*** at 59 to 61.)

With respect to the left hip condition, Dr. Friedman also concluded that Prednisone did not cause or aggravate it:

> A. Well, Prednisone does not cause arthritis. So my feeling is that it didn't aggravate it, and it didn't cause it. It was an independent factor.
>
> This has nothing to do with osteopenia or reduced calcium deposition in bone. This has to do with surface of a cartilage that it worn away between the acetabular surface and the head of the femur.

(***Id.*** at 26 to 28.) However, although he felt that more data would be necessary, he also admitted that Claimant had not had left hip problems prior to her treatment with Prednisone and he could not rule out the relationship between Prednisone and the left hip condition. (***Id.*** at 71, 75, 81.)

Upon consideration of the testimony of both of these physicians, I find that both hip conditions are compensable injuries. In this regard, I give more weight to the opinion of Dr. Wagner, Claimant's treating physician, because of his superior knowledge of the particulars of Claimant's treatment combined with Dr. Friedman's lack of knowledge as to the magnitude of Claimant's Prednisone dose. Moreover, although asserting that Prednisone does not cause osteoarthritis, Dr. Friedman has not really addressed the issue of whether the bone loss due to Prednisone resulted in either hip surgery being required at an earlier date. Based upon Dr. Wagner's testimony, I find that Claimant has established that, while it would have been advisable for the right hip replacement surgery to be eventually performed under any circumstances, it became necessary at an earlier time, and became no longer an elective procedure, due to her Prednisone treatment. The right hip condition was therefore aggravated and is compensable. I further find that Claimant has established that her left hip condition, and the need for a left hip replacement, was caused or aggravated by her Prednisone treatment. Both hip conditions are therefore compensable injuries.

Shingles (Herpes Zoster) and Ulnar Nerve Neuropathy. Both Drs. Wagner and Dr. Friedman opined that the Claimant's shingles, or herpes zoster, was caused by her Prednisone use, due to its impact on her immune system.[41] However, only Dr. Wagner found that the shingles resulted in Claimant's left ulnar [elbow] nerve neuropathy and the need for the March 2000 ulnar nerve surgery, and Dr. Friedman opined that it was due to trauma.[42] (***Compare*** CX K at 26 to 29 ***with*** CX J at 34 to 38, 159-157, 171-75). Dr. Wagner's testimony on this issue was equivocal, as he stated to a reasonable degree of medical certainty that the ulnar nerve problem was related to the shingles and that it was not related.(CX J at 37, 159). It became clear at the hearing that Dr. Wagner was not aware that the term reasonable medical certainty was intended to encompass a finding of "more probable than not." Upon closer examination, it is only the herpes zoster which Dr. Wagner has opined to be more probable than not the cause of the ulnar nerve disability, based upon the lack of prior ulnar nerve complaints and the distribution, although he acknowledged there were other possible etiologies. (CX J at 37 to 38, 154-59). Dr. Friedman pointed to the absence of nerve defects (except of the ulnar nerve) on the EMG as suggesting that trauma was

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[41] Dr. Wagner also opined that the Claimant's chronic pain syndrome and cellulitis resulted from the shingles. (CX J at 36 to 37.)

[42] Claimant's neurologist, Dr. Richard Edelson, stated in a September 21, 1999 Office Visit Follow-Up Note: ". . . The other problem has been the left ulnar atrophy. On the EMG it did show a relative block at the elbow. However, her shingles were higher and I think relate to a cervical nerve, but mainly affecting the ulnar innervation. Otherwise, the EMG did not show any evidence of a peripheral neuropathy. . ." (CX G3 at 927). The EMG report found "left ulnar neuropathy with relative block in the ulnar groove" but "no evidence to suggest a peripheral neuropathy." (***Id.*** at 929, 935-36.)

responsible. However, he also waffled somewhat on this issue, acknowledging that herpes zoster could cause fibrosis of the peripheral nerves and that he could not be sure that it was due to trauma, and he expressed uncertainty as to when she first experienced cervical problems and whether they could have been an early sign of shingles. (CX K at 66 to 71). Considering all of this evidence, I again find Dr. Wagner to have had superior knowledge of the particulars of Claimant's medical history and he has pointed to factors that make it more likely than not that Claimant's ulnar nerve problems were due to shingles, which was in turn due to Prednisone therapy. Accordingly, I find both the shingles/herpes zoster and the ulnar nerve neuropathy (necessitating the March 2000 surgery) are compensable injuries.

Coronary Artery Disease and Carbon Stent Implant. Dr. Wagner opined that the Claimant's coronary artery or cardiovascular disease (leading to angioplasty and carbon stent placement) was attributable in part to the Claimant's Prednisone treatment, by increasing Claimant's serum cholesterol levels and accelerating the atherosclerotic process. (CX J at 38 to 41, 162-63, 171). However, he acknowledged that he did not know what her cholesterol levels were at the time she had the heart stent, and he went on to testify that "there's probably a 50/50 chance without the cholesterol she would have ultimately had coronary artery disease" based upon the fact that 50 per cent of all women have coronary artery disease as some time in their lives . (***Id.*** at 163-64; ***see also id.*** at 170.) He also admitted that the increase in cholesterol was temporary. (***Id.*** at 165-66.) While acknowledging that other factors could have played a part, he maintained that the Prednisone aggravated the process. (***Id.*** at 175-76.) Dr. Friedman essentially agreed that the Prednisone may cause lipid abnormalities (including elevated cholesterol) and could have aggravated Claimant's coronary problems, but he noted that she had other risk factors, such as her smoking history. (CX K at 62-75).

In reviewing all of the evidence, I find that the possibility has been raised that the Claimant's Prednisone treatment caused or aggravated her coronary artery disease, thereby necessitating the carbon stent, by elevating Claimant's cholesterol and worsening plaque buildup. However, the evidence does not rise to the level of reasonable medical certainty. Even Claimant's treating physician, Dr. Wagner, has merely stated that there was a "50/50 chance" of such an association. Dr. Friedman also acknowledged the possibility but pointed to other risk factors. No cardiologist has expressed an opinion in this area. Thus, the hypothesis remains speculative, and I find that Claimant has not proven that her coronary artery disease and resulting carbon stent implant was in any way related to her employment.

Dental Problems. Dr. Wagner testified that Claimant's "fairly severe dental problems" were related to her Prednisone therapy as a result of the immunosuppressive effect of Prednisone. (CX J at 46 to 49, 151-52). Dr. Friedman indicated that he would not be capable of testifying as to the relationship between Prednisone and Claimant's ongoing dental problems. (CX K at 75 to 76). Although at the previous (October 27, 2000) hearing Employer conceded that the Claimant's dental problems were attributable to Prednisone therapy, Employer did not formally stipulate to that effect at either hearing before me. (TR1 at 33-34; TR2 at 10-11). Based upon

Dr. Wagner's unrefuted testimony, I find that Claimant's dental problems are compensable injuries.

Facial Disfigurement ("Moon Face"). Claimant testified at the initial hearing as to the significant changes in her physical appearance following Prednisone therapy and her development of the classic "moon face," for which she required plastic surgery. (See TR 1 at 74-75, CX I.). This and similar types of deformities are classic complications of high dose Prednisone therapy, as both Dr. Wagner and Dr. Friedman testified. (CX J at 43-46; CX K at 62-66). It is undisputed that Claimant's facial disfigurement or "moon face" is attributable to her Prednisone therapy, even though there was no formal stipulation to that effect. (TR1 at 33-34, TR2 at 10-11). I find that Claimant's facial disfigurement ("moon face") is a compensable injury and that the plastic surgery for its correction is a compensable medical expense.

Conclusion

In conclusion, I find that there is a basis for modification based upon a mistake in determination of fact on the rebuttal issue, in that the rebuttal was inadequate to sever the causal connection between Claimant's employment in Almaty and her pulmonary fibrosis, but that even if the rebuttal were deemed to be adequate, there was a mistake in determination of fact on the causation issue overall. On consideration of the merits of this claim, I find that the Claimant has established that her pulmonary condition presumptively arose out of her employment in Almaty, that the rebuttal adduced by the Employer is insufficient to sever the causal relationship, and that even if the rebuttal were deemed to be adequate, Claimant has established that her pulmonary condition arose out of her exposure to environmental pollutants during the course of her employment in Almaty.

I also find that Claimant has established that her right and left hip conditions, shingles (herpes zoster), ulnar nerve neuropathy, facial disfigurement ("moon face"), and dental tooth decay and disease were related to or aggravated by either the pulmonary fibrosis or Prednisone treatment for the pulmonary fibrosis, but that she has failed to establish that her coronary artery disease and carbon stent implant were so related or aggravated.

I further find that Claimant is entitled to medical benefits based upon her expenses for the treatment of these conditions (with the exception of coronary artery disease and carbon stent implant). In CX H1, she has itemized expenses of $166,958.77 for the period from May 1998 through May 2000. Claimant shall not be entitled to reimbursement for the amounts attributable to the stent implant. The roster at pages 1375 to 1377 of CX H1 itemizes these expenses from May 14, 2000 to May 29, 2000 in the amount of $13,040.21, leaving a compensable amount of $153,918.56 through May 2000, plus any later medical expenses, including those relating to the left hip replacement.

I also find that Claimant is entitled to compensation for the following periods of temporary total disability (TTD): June 6, 1998 to February 1, 1999 for pulmonary fibrosis and partial lung

biopsy, and for right hip surgery; March 3, 2000 to March 21, 2000 for left ulnar nerve surgery; and October 27, 2000 to June 1, 2001 for left hip replacement surgery.

In view of the above,

ORDER

IT IS HEREBY ORDERED that the Claimant's request for modification and claim for benefits is **GRANTED**, to the extent set forth above; and

IT IS FURTHER ORDERED that (1) Claimant is entitled to compensation and medical expenses relating to her pulmonary fibrosis, right and left hip conditions, shingles (herpes zoster), ulnar nerve neuropathy, facial disfigurement ("moon face"), and dental tooth decay and disease; and (2) Claimant is entitled to temporary total disability (TTD) payable at the maximum rate for the following periods: June 6, 1998 to February 1, 1999; March 3, 2000 to March 21, 2000; and October 27, 2000 to June 1, 2001.

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PAMELA LAKES WOOD
Administrative Law Judge

Washington, D.C.

Date: February 28, 2003